

REMARKS

The requisite fee for filing the Request for Continued Examination, the fee for a three-month extension of time and any other fees that may be due in connection with the filing of this paper or with this application should be charged to Deposit Account No. 02-1818. If a Petition for extension of time is needed, this paper is to be considered such Petition. A Supplemental Information Disclosure Statement and a second executed Declaration of Dr. Thomas J. Borody, pursuant to 37 C.F.R. §1.132, accompany this response.

Claims 1-11 and 37-41 are pending. Claims 40 and 41 are added and claims 12-18 and 34-36, directed to non-elected subject matter, are cancelled without prejudice or disclaimer. Applicant reserves the right to file divisional/continuing applications to any cancelled or unclaimed subject matter. The specification is amended to correct an inadvertent typographical error. Mannitol and lactulose inadvertently were included in a list of degradable sugars. Mannitol is not metabolized to any appreciable extent and is minimally reabsorbed in the body and thus is a minimally degradable sugar, not a degradable sugar (see, *e.g.*, *Remington Pharmaceutical Sciences* (16th edition, Osol, ed., 1980), page 873). Lactulose is not metabolized and is minimally absorbed in the body and thus is a minimally degradable sugar, not a degradable sugar (Carulli *et al.*, *Digestion* 6:139-145 (1972), Abstract). Claims 1, 8 and 9 are amended for clarity. Basis for claims 40 and 41 is found at page 10, lines 16-19. No new matter has been added.

Information Disclosure Statement

When submitting Information Disclosure Statements (IDSs), the undersigned includes a transmittal letter, a paper providing information, and, when appropriate, a Form PTO-1449. In some instances, the paper providing information includes a table with documents that require initials by the Examiner. The IDS provided herewith includes a table of documents to be reviewed that requires initials by the Examiner. The Table is provided on a paper that identifies the document as an Information Disclosure Statement and includes the serial number and other identifying information on every page. Applicant respectfully requests that the Examiner consider and initial the information listed in the Table.

REJECTION OF CLAIMS 1-11 AND 37-39 UNDER 35 U.S.C. 103(a)

Claims 1-11 and 37-39 are rejected under 35 U.S.C. 103 (a) as unpatentable over Kawakami (JP 05306221) in view of Colliopoulos (US 5,232,699) in view of Cockerill (US 4,452,779) because Kawakami allegedly teaches every element of the claims except xylose as a minimally degradable sugar, magnesium sulfate as a water-soluble magnesium salt and a hypertonic aqueous solution, but Colliopoulos and Cockerill allegedly teach the

elements missing from Kawakami. The Examiner alleges that it would have been obvious to one of ordinary skill in the art to have combined the teachings of Kawakami, Colliopoulos and Cockerill and to have added xylose as a minimally degradable sugar in the composition of Kawakami and to use magnesium sulfate as a water-soluble magnesium salt in a hypertonic solution (claim 8). The Examiner contends that the selection of the weight ratios is merely a matter of judicious selection and routine optimization. In maintaining the rejection, the Examiner alleges that:

Kawakami teaches the saccharide contains sugar types having sweetness, for instance, sucrose, maltose, grape sugar, invert sugar, and the like. Because Kawakami teach that sugar types having sweetness can be used it would have been obvious to the skilled artisan to try any one of the sweeteners taught in the Colliopoulos reference, including xylose, glucose and fructose. It would have been obvious to the skilled artisan to use either of the sweetening agents as these are common sweetening agents used in laxative compositions as evidenced by Moskowitz, US Patent No. 4,766,004, which is incorporated by reference in Colliopoulos, and Andre *et al.*, US Patent No. 5,173,296. Moskowitz teaches that sweetening agent ingredients used in the compositions include water-soluble sweetening agents such as monosaccharides such as xylose, ribose, glucose, fructose and sucrose (col. 5, lines 53-56). Andre *et al.* teach compositions that provide laxation and regulating bowel function (col. 3, lines 7-12). Andre *et al.* teach the compositions comprise a sweetening agent that includes water-soluble sweetening agents such as xylose, ribose, glucose, fructose and sucrose. As such, the skilled artisan would have been motivated at the time of the invention to make the substitution because as evidenced by the prior art, these sweeteners are known to be used in [*sic*] as sweetening agents in laxative compositions.

The arguments in the previous response are incorporated herein by reference.

Reconsideration of the grounds for this rejection respectfully is requested in view of the following remarks.

ANALYSIS

It respectfully is submitted that the Examiner has failed to set forth a case of *prima facie* obviousness for the following reasons.

The combination of the teachings of Kawakami and Colliopoulos and Cockerill does not result in the claimed compositions and methods

Claim 1

The purgative composition of claim 1 contains (i) at least one water-soluble sodium salt; (ii) at least one water-soluble minimally degradable sugar, where the total weight of water-soluble minimally degradable sugar in the composition is from about 1 to about 3 times the weight of sodium salt in the composition; (iii) at least one water-soluble potassium salt, where the weight of the water-soluble potassium salt in the composition is from about 0.05 to about 1 times the weight of the sodium salt in the composition; and (iv) at least one water-soluble magnesium salt, where the weight of magnesium salt in the composition is from about 0.1 to about 10 times the weight of sodium salt in the composition.

The composition taught by Kawakami, when dissolved in 900 mL of water as described in Kawakami, yields an aqueous solution that includes 32.3 to 35.7 grams of magnesium citrate, 4.8 to 5.4 mmol sodium chloride, 8.5 to 9.3 mmol potassium hydrate [potassium hydroxide] and 2.1 to 10.7 grams degradable sugars (paragraphs [0014] and [0017]). Thus, the composition described in Kawakami, before it is dissolved in 900 mL of water, includes 0.25-0.29 grams sodium chloride, 2.1 to 10.7 grams degradable sugar (7.3× to 42.8× the amount of sodium salt), 0.43-0.47 grams potassium hydroxide (1.48× to 1.88× the amount of sodium salt), and 32.3-35.7 grams magnesium citrate (111.4× to 142.8× the amount of sodium salt). Kawakami teaches that exemplary sugars in its formulation include sucrose, maltose, grape sugar, fructose and invert sugar (paragraph [0016]). Thus, the composition described in Kawakami includes a degradable sugar in an amount that is 7.3× to 42.8× the amount of sodium salt. Kawakami teaches that it is the combination of magnesium citrate, sodium chloride, potassium hydroxide and a degradable sugar such as sucrose in the recited fixed range and ratio that allows its composition to function as a purgative without causing metabolic electrolyte problems (paragraphs [0012] and [0013]). Kawakami teaches that exemplary sugars in its formulation include sucrose, maltose, grape sugar, fructose and invert sugar (paragraph [0016]). None of these is a minimally degradable sugar. None is resistant to endogenous digestion in the gastrointestinal tract.

Kawakami teaches that mannitol, which is a minimally degradable sugar, is not used in its compositions because the presence of mannitol leads to the production of hydrogen and methane gas due to the decomposition of the mannitol by bacteria in the intestine, and this gas could lead to an accidental explosion of the intestine during operations (paragraph [0005]). Thus, Kawakami *et al.* fails to teach or suggest, among other elements, a purgative composition that contains a minimally degradable sugar at a ratio 1 to about 3 times the weight of sodium salt in the composition. None of the secondary references teaches or suggests the elements missing from Kawakami.

Colliopoulos teaches baked wafer laxative compositions containing psyllium and senna or sennosides dispersed in a food grade fat and 5% to about 40% of a sweetening agent. Colliopoulos provides a long list of sweetening agents, including xylose, ribose, glucose, mannose, galactose, fructose, dextrose, sucrose, maltose, partially hydrolyzed starch or corn syrup solids, sorbitol, xylitol, mannitol, sodium or calcium saccharin salts, cyclamate salts, acesulfame-K, the free acid form of saccharin; or dipeptide based sweeteners such as L-aspartyl-L-phenylalanine methyl ester and materials described in U.S. Pat. No. 3,492,131 (see. col. 6, lines 51-61). There is no teaching or suggestion in Colliopoulos to add a

minimally degradable sugar to a purgative composition of Kawakami, nor for adding a minimally degradable sugar in an amount from about 1 to about 3 times the weight of sodium salt in the composition.

Moskowitz, cited for its teaching of sweeteners, teaches a dietary fiber supplement composition that contains whole psyllium husks. The composition of Moskowitz is similar to the composition of Colliopoulos, and Moskowitz teaches a laundry list of sweeteners that may be used in its wafer. There is no teaching or suggestion in Moskowitz for selecting a minimally degradable sugar as a sweetener, nor for adding a minimally degradable sugar to that purgative composition of Kawakami, nor for adding a minimally degradable sugar in an amount from about 1 to about 3 times the weight of sodium salt in the composition as instantly claimed.

Andre *et al.*, cited for its teaching on sweeteners, teaches a marzipan-like composition that contains psyllium fiber for use as a laxative. The composition of Andre *et al.* is similar to the compositions of Colliopoulos and Moskowitz, and Andre *et al.* teaches a laundry list of sweeteners that may be used in its composition. There is no teaching or suggestion in Andre *et al.* for selecting a minimally degradable sugar as a sweetener, nor for adding a minimally degradable sugar to the purgative composition of Kawakami, nor for adding a minimally degradable sugar in an amount from about 1 to about 3 times the weight of sodium salt in.

The compositions of Cockerill do not include a minimally degradable sugar nor any sugar. Cockerill does not teach or suggest including a minimally degradable sugar, such as xylose, in any formula. There is no teaching or suggestion whatsoever with respect to sugar in Cockerill, and the reference certainly does not teach or suggest adding a minimally degradable sugar to a purgative composition nor any composition that includes a minimally degradable sugar in an amount from about 1 to about 3 times the weight of sodium salt in the composition as instantly claimed.

None of Colliopoulos, Cockerill, Moskowitz or Andre *et al.* teaches or suggests any advantageous property or any reason for including a minimally degradable sugar, nor including it in an amount from about 1 to about 3 times the weight of sodium salt. Kawakami specifically teaches that minimally degradable sugars should not be included in its composition. Therefore, in view of the teachings of Kawakami that minimally degradable sugars, such as mannitol, should not be included in purgative formulations because of the potential of explosive gas formation, and because none of Colliopoulos, Cockerill, Moskowitz or Andre *et al.* teaches or suggests anything with respect to minimally degradable sugars other than including such sugars in a laundry list of sweeteners, one of ordinary skill

in the art could **not** have selected a minimally degradable sugar from the list of sweeteners in Colliopoulos or Moskowitz or Andre *et al.* for use in a purgative as described in Kawakami nor to include it in a ratio of about 1× to about 3× the weight of sodium salt. Kawakami teaches inclusion of a degradable sugar at a ratio of from 7.3× to 42.8× the weight of sodium salt. According to the teachings of Kawakami, use of a minimally degradable sugar such as mannitol would result in a composition that would produce explosive gases when used as a purgative. Thus, in light of the teachings of Kawakami, the modification of the Kawakami composition proposed by the Examiner would render it unsatisfactory, due to the alleged undesirable side-effect taught by Kawakami. Thus, there can be no suggestion or motivation to have made the proposed modification (*In re Gordon*, 733 F.2d 900, 221 USPQ 1125 (Fed. Cir. 1984)), since such modification is taught by Kawakami to be undesirable. Therefore, teachings of the references cannot render the claims *prima facie* obvious. *In re Ratti*, 270 F.2d 810, 123 USPQ 349 (CCPA 1959).

Further, none of the cited art nor any combination thereof teaches or suggests a purgative composition as instantly claimed having the recited ratio of components. A 900 mL dosage of the composition described in Kawakami includes 0.25-0.29 grams sodium chloride, 2.1 to 10.7 grams degradable sugar (7.3× to 42.8× the amount of sodium salt), 0.43-0.47 grams potassium hydroxide (1.48× to 1.88× the amount of sodium salt), and 32.3-35.7 grams magnesium citrate (111.4× to 142.8× the amount of sodium salt). When set forth as based on the weight of the sodium salt in the composition, it is apparent that the instantly claimed compositions are significantly different from the compositions of Kawakami.

Components based on the amount of sodium salt in the composition

Component	Instant Compositions	Kawakami compositions
minimally degradable sugar	1× to 3×	--
degradable sugar	--	7.3× to 42.8×
Potassium salt	1.48× to 1.88×	0.05× to 1×
Magnesium salt	111.4× to 142.8×	0.1× to 10×

None of Colliopoulos, Cockerill, Moskowitz or Andre *et al.*, alone or in any combination, teaches or suggests a composition that contains, or would lead one of ordinary skill in the art to modify the composition of Kawakami so that it contains, a minimally degradable sugar in an amount that is about 1 to about 3 times the weight of sodium salt in

the composition; and contains an amount of water soluble potassium salt in an amount that is about 0.05 to about 1 times the weight of the sodium salt in the composition; and contains an amount of water soluble magnesium salt in an amount that is about 0.1 to about 10 times the weight of sodium salt in the composition. Therefore, for at least these reasons, the combination of the teachings of Kawakami and Colliopoulos and Cockerill does not teach or suggest every element of the compositions of claims 1-7 and 37-39.

Claims 8 and 9

Claim 8 recites a purgative composition of claim 1 in the form of a hypertonic aqueous solution. Claim 9 depends from claim 8 and includes every limitation thereof. Kawakami teaches that its composition is dissolved in 900 mL of water to provide a purgative that is isotonic (paragraphs [0017] and [0018]). Kawakami teaches that it is important to provide an isosmolar solution because it is only products having an osmotic pressure range of 290 through 310 mOsmol/L that do not produce water absorption into the body or water removal from the body in the intestinal canal and thus avoids changes in electrolyte balance (paragraphs [0018] and [0012]). Kawakami teaches that isotonic compositions are necessary to avoid metabolic electrolyte problems. Kawakami teaches that it is the recited fixed range and ratio of magnesium citrate, sodium chloride, potassium hydroxide and a degradable sugar such as sucrose that allows its composition to function as a purgative without causing metabolic electrolyte problems. There is no teaching or suggestion in Kawakami to provide a hypertonic aqueous composition as claimed in claim 8.

Neither Colliopoulos nor Cockerill teaches or suggests a hypertonic aqueous solution. Colliopoulos teaches a baked wafer laxative composition that contains psyllium and senna. Colliopoulos does not teach or suggest any solutions and certainly does not teach or such modifying an isosmolar solution to make it hyperosmolar. Cockerill does not teach or suggest the elements missing from the combination of Kawakami and Colliopoulos. Cockerill teaches dry powdered compositions that are mixed with the feed of a lactating mammal. The only mention of a hypertonic solution in Cockerill is that the saline cathartic in its formulation forms a hypertonic solution in the intestine and that this formation in the intestine sets up an osmotic pressure differential driving water into the intestine (see col. 2, lines 19-37). There is no mention, teaching or suggestion of a purgative in the form of an aqueous hypertonic solution in Cockerill. Thus, combining the teachings of Kawakami and Colliopoulos and Cockerill, alone or in any combination, does not teach or suggest an aqueous hypertonic solution as recited in claims 8 and 9. Therefore, the combination of the teachings of Kawakami and Colliopoulos and Cockerill does not teach or suggest every element of claims 8 and 9.

In addition, according to the teachings of Kawakami, use of a hypertonic solution would result in metabolic electrolyte problems. Kawakami teaches that it is necessary to formulate the product to have an osmotic pressure range of 290 through 310 mOsmol/L to prevent water absorption into the body or water removal from the body in the intestinal canal in order to avoid changes in electrolyte balance. Thus, in light of the teachings of Kawakami, the modification of the Kawakami composition proposed by the Examiner to make the composition hypertonic would render it unsatisfactory, due to the alleged undesirable side-effects taught by Kawakami. Thus, there can be no suggestion or motivation to have made the proposed modification (*In re Gordon*, 733 F.2d 900, 221 USPQ 1125 (Fed. Cir. 1984)), since such modification is taught by Kawakami to be undesirable. Therefore, teachings of the references cannot render the claims *prima facie* obvious. *In re Ratti*, 270 F.2d 810, 123 USPQ 349 (CCPA 1959).

DECLARATION

Notwithstanding the above, the attached Declaration of Dr. Borody demonstrates results not taught or suggested by the cited art.

The instantly claimed compositions of claim 1 can be administered, *e.g.*, in capsules (see claim 7), can be dissolved in water to provide an aqueous hypertonic solution (as recited in claim 8) or can be administered in other forms. The composition of Kawakami is dissolved in 900 mL of water to provide an isotonic magnesium citrate-based purgative.

Dr. Borody, a joint inventor of the claimed subject matter, has compared the compositions of claim 1, administered in the form of capsules, to a commercially available magnesium citrate-based purgative that is sold under the trade name PicoPrep™. Dr. Borody states that PicoPrep™ is similar to the magnesium citrate-based purgative described in Kawakami. Dr. Borody states that, although not recommended, when PicoPrep™ is administered with a fruit juice or other beverage that contains a degradable sugar, the PicoPrep™ preparation then includes a degradable sugar as recited in the composition of Kawakami and, for purposes of the DECLARATION, is comparable to the aqueous purgative composition of Kawakami because it includes a degradable sugar such as sucrose or fructose (*e.g.*, see Dennison, *J Am College of Nutrition* 15(5): 4S-11S (1996); and Duthie, *NutriDate* 18(1): 5-8 (2007)).

The attached DECLARATION of Dr. Borody describes clinical data generated from the comparison testing of the compositions of claim 1 administered in capsules to the aqueous magnesium citrate-based purgative PicoPrep™ with and without degradable sugar.

Bowel cleansing

A composition within the scope of the instant claim 1 and its dependent claims administered in capsules, which is referred to in the DECLARATION as "Hydroprep," was more effective in cleansing the bowel than the PicoPrep™ purgative administered with a degradable sugar or in the absence of a degradable sugar. The adequacy of bowel wall cleansing was judged by two observers and documented by photography. As discussed in the DECLARATION, the Hydroprep composition was judged to provide significantly better bowel wall cleansing than achieved using the PicoPrep™ composition, regardless of whether the PicoPrep™ purgative was administered with or without a degradable sugar, and this enhanced cleaning was achieved in less time. Although various combinations of weight ratios of salts and sugars were tested, it was only when a composition falling within the scope of the instant claim 1 was used that there was such a dramatic improvement in bowel cleansing. None of the cited art or any art of record teaches or suggests that a combination of water soluble sodium, potassium and magnesium salts and minimally degradable sugar(s) in the recited ratios results in a composition that is more effective in bowel cleansing than existing purgatives. The weight ratios of the water soluble salts and minimally degradable sugar(s) in the claimed compositions of claim 1 administered in capsules results in enhanced purgative properties not taught or suggested in the prior art.

The enhanced cleansing of the instant compositions of claim 1 and its dependent claims administered in capsules was observed when the instant compositions contained different minimally degraded sugars. As discussed in the DECLARATION, the Hydroprep composition containing the minimally degradable sugar xylose exhibited an even more enhanced bowel cleansing than exhibited by the Hydroprep composition containing mannitol. Inulin, also a minimally degradable sugar (see, *e.g.*, Ellegård *et al.*, European J Clinical Nutrition 51:1-5 (1997), which teaches on page 4 that only minor hydrolysis or bacterial degradation of inulin occurs during intestinal passage), also demonstrated enhanced purgative properties similar to those observed with mannitol. Thus, purgative compositions within the scope of the instant claim 1 and its dependent claims administered in capsules that include minimally degradable sugars of various classes, including monosaccharide (xylose), oligosaccharide (inulin) and sugar alcohols (mannitol), exhibit enhanced bowel cleansing compared to PicoPrep™ administered with or without degradable sugar.

Reduced time to first defecation

The DECLARATION also states that notable acceleration of time to initiating bowel evacuation was observed with compositions within the scope of claim 1 and its dependent

claims administered in capsules, compared to purgatives such as the PicoPrepTM composition and the composition of Kawakami. First defecation in patients administered the Hydroprep composition occurred between 2 and 4 hours after administration. In contrast, first defecation in patients administered the PicoPrepTM composition, with or without degradable sugar, occurred between 3 and 5 hours after administration. Thus, a more rapid onset of cleansing was observed with the Hydroprep composition than with purgatives such as the PicoPrepTM composition and the composition of Kawakami, which do not include a minimally degradable sugar in an amount that is about 1 to about 3 times the weight of sodium salt in the composition.

Side effects

Also, as discussed in the specification and the DECLARATION, when administered to patients, the instantly claimed compositions resulted in fewer adverse side effects than PicoPrepTM. As discussed in the DECLARATION, when the occurrence of adverse side effects in the patient groups was compared, patients administered the Hydroprep composition experienced a 92% reduction in nausea, 100% reduction in vomiting and a 37% reduction in headaches as compared to patients treated with the PicoPrepTM composition.

Conclusion

The Declaration of Dr. Borody demonstrates that purgatives that are within the scope of claim 1 and its dependent claims are effective at cleansing of the bowel and, are superior in cleansing ability to the PicoPrepTM composition. Since the PicoPrepTM composition is comparable to the composition described in Kawakami, Dr. Borody infers that the instantly claimed compositions are superior to the composition of Kawakami. The cleansing occurs in less time with the Hydroprep composition than the time required for first defecation in patients administered the PicoPrepTM composition. The Hydroprep composition, which includes a minimally degradable sugar, causes fewer adverse side effects, such as nausea, vomiting and headaches, than are caused by purgatives such as the PicoPrepTM composition and the composition described in Kawakami, which do not include a minimally degradable sugar in an amount that is about 1 to about 3 times the weight of sodium salt in the composition.

None of Kawakami or Colliopoulos or Cockerill, singly or in any combination, nor any art of record, teaches or suggests any combination of water soluble sodium, potassium and magnesium salts and minimally degradable sugar(s) in the recited weight ratios or that such a combination would result in a composition that exhibits superior bowel cleansing than is achieved using the PicoPrepTM composition, which is comparable to the composition

described in Kawakami. None of the references, alone or in any combination, teaches or suggests that a purgative composition as recited in claim 1 and its dependent claims is more effective at cleansing the bowel. None of the references, alone or in any combination, teaches or suggests that a composition of claim 1 and its dependent claims would initiate bowel cleansing faster or would exhibit fewer adverse side effects than the PicoPrep™ composition with or without degradable sugar. As discussed in the DECLARATION, although various combinations of weight ratios of salts and sugars were tested, it was only when compositions falling within the scope of claim 1 and its dependent claims were used that these benefits (a significant improvement in bowel cleansing, a faster time to initiation of bowel cleansing, and fewer adverse side effects) were achieved. None of Kawakami or Colliopoulos or Cockerill, singly or in any combination, teaches or suggests that the instantly claimed ratios and ingredients produce such results. Therefore, the claims cannot be obvious in view of the cited references.

REBUTTAL TO EXAMINER'S ARGUMENTS

1. SUGAR REPLACEMENT

Applicant respectfully submits that replacing the sucrose or other degradable sugar in the composition of Kawakami with xylose "at the same ratios" as suggested by the Examiner does not result in the instantly claimed compositions. The instant compositions include water-soluble minimally degradable sugar in an amount from about 1 to about 3 times the weight of sodium salt in the composition. The composition described in Kawakami is a 900 mL solution that includes 4.8-5.4 mM sodium chloride, 8.5-9.3 mM potassium hydrate and 10.7-2.1 grams sugar. The ratio of sugar to sodium salt in the compositions of Kawakami is more than twice the upper recited limit of the minimally degradable sugar in the instant compositions. Therefore, even if one of ordinary skill in the art would have replaced the sucrose or degradable sugar in the composition of Kawakami with xylose "at the same ratios" as suggested by the Examiner, this does not result in the instantly claimed compositions.

In addition, if one of ordinary skill in the art were selecting a sweetening agent merely for its ability to sweeten, Applicant respectfully submits that one would not use an equivalent amount of xylose to provide the equivalent sweetness achieved by sucrose or fructose, degradable sugars that can be used in the compositions of Kawakami. Xylose only has a sweetness value of 40 when compared to sucrose, which has a sweetness value of 100 (see Booth *et al.*, Journal of Nutrition 49: 347-355 (1953)). Thus, it would take more than twice the amount of xylose to provide an equivalent sweetness obtained if the degradable sugar in the compositions of Kawakami was sucrose. At this usage level of xylose, the ratio of sugar

to sodium salt in the compositions of Kawakami would be more than four times the upper recited limit of the minimally degradable sugar in the instant compositions. Fructose has a sweetness value of 120-170 (*Alternative Sweeteners* (O'Brien Nabors, ed. (2001), page 3). Thus, it would take more than three to four times the amount of xylose to provide an equivalent sweetness obtained if the degradable sugar in the compositions of Kawakami was fructose. At this usage level of xylose, the ratio of sugar to sodium salt in the compositions of Kawakami would be more than six to eight times the upper recited limit of the minimally degradable sugar in the instant compositions. Thus, routine optimization and selection of a minimally degradable sugar would not have resulted in the instantly claimed compositions.

Furthermore, as discussed above, the combination of teachings of the cited references, singly or in any combination, does not teach or suggest the results achieved with the instantly claimed compositions. Unexpected properties (properties not taught or suggested by the cited art) must always be considered in establishing *prima facie* obviousness.

2. NOT ROUTINE OPTIMIZATION

In maintaining the previous rejection under 35 U.S.C. §103(a), the Examiner states that the instantly claimed compositions are obvious because they can be obtained by mere optimization of known components. Applicant respectfully disagrees. Kawakami teaches that mannitol, which is a minimally degradable sugar, is not used in purgative compositions due to the hydrogen and methane gas produced by the decomposition of the mannitol by the bacteria in the intestines and the possible explosion of the intestines during operations due to these gases. Thus, routine optimization would not lead one of ordinary skill in the art to include a minimally degradable sugar in a purgative composition, particularly in light of the teachings of Kawakami not to include a minimally degradable sugar because of possible explosion of the intestines during operations. In addition, as note above, if a minimally degradable sugar were used as a sweetener as suggested by the Examiner, routine optimization would not have resulted in the instantly claimed ratios, since far more minimally degradable sugar would have been required to achieve the necessary sweetness.

Further, with respect to claims 8 and 9, Kawakami teaches that its composition is an isosmolar magnesium citrate solution that includes sodium chloride, potassium hydroxide and a degradable sugar such as sucrose. Kawakami teaches that its composition is effective as a purgative because it is optimized to be isosmolar to provide intestinal canal irrigation while minimizing problems of electrolytic metabolism because its composition does not result absorption of water from the composition or absorption of water from the intestinal canal by

the composition. There is no evidence provided by the Examiner that the type and ratio and amount of salts and degradable sugar in the isosmolar composition of Kawakami, or any teaching of Colliopoulos or Cockerill, or any combination thereof, would lead one of ordinary skill in the art to formulate a hyperosmolar composition in which the amount and ratio of salts is modified and a minimally degradable sugar is included. None of the cited art teaches or suggests modifying a magnesium citrate-based purgative such as that described in Kawakami by using about one quarter the amount of magnesium salt, about 7.5 times the amount of potassium salt and about 16 times the amount of sodium salt. The fact that one of ordinary skill in the art *could* reformulate the compositions of Kawakami to be a hyperosmolar dosage, which is significantly different from the recommended isosmolar dosage of Kawakami, and *could* select different salts in differing ratios, does not provide any reason to do so. The cited art must provide a reason to do that which applicant has done. The mere fact that prior art may be modified to produce the claimed product does not make the modification obvious unless the prior art suggests the desirability of the modification. In re Fritch, 23 U.S.P.Q.2d 1780 (Fed. Cir. 1992); see, also, In re Papesch, 315 F.2d 381, 137 U.S.P.Q. 43 (CCPA 1963). None of Kawakami, Colliopoulos or Cockerill or any combination thereof teaches or suggests a composition containing a combination of water soluble sodium, potassium and magnesium salts and minimally degradable sugar(s) in ratios as instantly claimed nor provides or suggests any reason to modify the magnesium citrate-based purgative of Kawakami to be a combination of water soluble sodium, potassium and magnesium salts in combination with a minimally degradable sugar as instantly claimed.

* * *

In view of the remarks herein, reconsideration and allowance respectfully are requested.

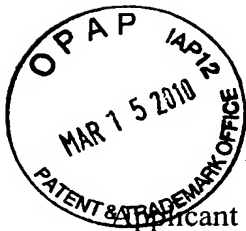
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Commissioner for Patents

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ATTACHMENTS

1. *Remington Pharmaceutical Sciences* (16th edition, Osol, ed., 1980), page 873.
2. Carulli *et al.*, *Digestion* 6:139-145 (1972), Abstract.
3. Booth *et al.*, *Journal of Nutrition* 49: 347-355 (1953).
4. *Alternative Sweeteners* (O'Brien Nabors, ed. (2001), page 3.
5. Ellegård *et al.*, *European J Clinical Nutrition* 51:1-5 (1997).
6. Dennison, *J Am College of Nutrition* 15(5): 4S-11S (1996)
7. Duthie, *NutriDate* 18(1): 5-8 (2007)

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Remington's

ARTHUR OSOL

*Editor, and Chairman
of the Editorial Board*

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Chapter 49

Diuretic Drugs

osmotic diuretics
renal tubular
inhibiting
diuretics
miscellaneous
renal agents

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Diuretics are drugs used to increase the volume of urine excreted by the kidneys. They are employed principally for the relief of edema and ascites. These conditions occur in diseases of the heart, kidneys, and liver. Diuretics are most effective in the treatment of cardiac edema, particularly that associated with congestive heart failure. They are also used in the ascites of cirrhosis, the nephrotic syndrome, diabetes insipidus, hypertension, edema of pregnancy, and to reduce cerebrospinal and intraocular fluid pressure. Some diuretics have highly specialized uses in glaucoma, hyperkalemia, bromide intoxication, anginal syndrome, epilepsy, migraine, hypertension, and in premenstrual depression, conditions in which edema is not present or at least not definitely established.

The formation of urine from the blood, in simplest terms, consists of glomerular filtration and selective tubular reabsorption and secretion. As the glomerular filtrate passes through the tubules, substances essential to the blood and tissues—water, glucose, salts, and amino acids—are reabsorbed. Other substances in the glomerular filtrate, such as urea, are not as readily absorbed by the tubules. Thus, it is thought that in the renal tubule there is a specific mechanism for the transport of each ionic species, the capacities of which are quite different. For example, the capacity of the renal tubule to reabsorb sulfate ion is limited. The tubular capacity for the reabsorption of phosphate is such that sufficient is reabsorbed to maintain the normal extracellular level, and any excess is excreted. On the other hand, much larger amounts of bicarbonate ion and chloride ion can be reabsorbed.

Under normal circumstances the glomerular filtration rate is about 100 ml/min. About 99 ml of the fluid is returned to the blood and only 1 ml is excreted as urine. It follows, therefore, that drugs may increase the rate of urine formation in two ways: (1) by increasing glomerular filtration and (2) by depressing tubular reabsorption. Increasing glomerular filtration is not an efficient mechanism and usually causes only a moderate increase in urine formation. If, for example, the percent of fluid reabsorbed by the renal tubules is assumed to remain constant, glomerular filtration rate would have to be increased twofold in order to double the urinary output. On the other hand, a 1% decrease in the tubular reabsorption of water, induced either by the administration of excessive quantities of electrolytes or nonelectrolytes (osmotic diuretics) or by agents which alter selective reabsorption of substances in the renal tubules, would double the urinary output.

Most diuretics block sodium and/or chloride reabsorption in the renal tubules. This results in natriuresis and diuresis. However, the mechanism(s) by which diuretics block the reabsorption and the site of action varies; they may act at either the proximal tubule, loop of Henle, distal tubule, or combinations of these sites. Agents which act at the proximal tubule include the osmotic diuretics (mannitol), carbonic anhydrase inhibitors (acetazolamide), and the organic mercurial diuretics (mercaptomerin sodium). Mannitol is not metabolized to any appreciable extent and is minimally reabsorbed when presented to the renal tubule; hence the

hyperosmolarity of the glomerular filtrate limits water reabsorption in the proximal tubule and results in diuresis with an increased potassium loss. The carbonic anhydrase inhibitors act on the proximal convoluted, via the intermediate step of adenylyl cyclase inhibition; to decrease bicarbonate reabsorption and passive forces favoring chloride reabsorption. The organic mercurials act on the proximal tubule and the first portion of the distal tubule by liberating mercuric ions which combine with sulfhydryl receptors to promote excretion of sodium as the chloride. Furosemide acts mainly on the ascending limb of the loop of Henle, although it is also thought to have some effect on both the proximal and distal tubules. Since furosemide also induces significant kaliuresis, supplemental administration of potassium is often necessary. Ethacrynic acid acts at similar sites; i.e., ascending loop of Henle plus the proximal and distal tubules. Action at these multiple sites is thought to account for its enhanced potency over thiazides. Since ethacrynic acid induces a greater excretion of chloride than sodium, it can produce systemic alkalosis. Ethacrynic acid, in contrast to the mercurials, continues to be effective in the presence of alkalosis. It is also useful in cases of edema refractory to other drugs. Thiazide diuretics act mainly to block sodium and chloride reabsorption at the first (thick) portion of the distal tubule. They also have a mild anti-carbonic anhydrase effect. The resulting natriuresis is accompanied by increased excretion of potassium, bicarbonate, chloride, and water. Unlike carbonic anhydrase inhibitors and organic mercurials, thiazide diuretics are effective even though systemic acidosis or alkalosis may be present.

The antihypertensive action of the thiazides is attributable to two factors: (1) depletion of sodium and subsequent reduction in plasma volume and (2) a decrease in peripheral resistance. The latter is thought to be due either to the loss of sodium from the arteriolar wall or a direct action on the vascular bed. In addition, there is some inhibition of the pressor activity of norepinephrine. In contrast, the antihypertensive effect of chlorthalidone is thought to be due to a decreased cardiac output.

Spironolactone, a potassium-sparing agent, blocks aldosterone by competitive inhibition at receptor sites in the collecting duct and not the distal portion of the tubule as previously presumed. This results in decreased excretion of potassium and decreased reabsorption of sodium and chloride accompanied by increased excretion of water. Diuresis can be markedly enhanced by concomitant administration of a thiazide. In addition, the onset of diuresis with combination therapy is much more rapid than with spironolactone alone (4 to 7 days). Triamterene acts to block sodium reabsorption and potassium excretion at that segment of the collecting duct under the control of adrenal mineralocorticoids. This is a direct effect and is unrelated to the level of aldosterone excretion.

Carbonic anhydrase inhibitors, such as acetazolamide, decrease the concentration of hydrogen ions available for exchange with sodium and potassium and to combine with

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Current

*Paper***Absorption of Lactulose in Man**

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**Abstract**

Lactulose absorption was studied in 6 normal subjects after the administration of different doses of lactulose. Lactulose was detected in the blood of only one patient. No changes were observed in the blood levels of glucose, galactose or lactate during the 24 h of the study in any of the volunteers. The amount of lactulose excreted in the urine was less than 1 % of the administered dose in 5 subjects and 2.8% in the 6th. Lactulose was detectable in urine 2 h after ingestion. At 8 h, the excretion was almost completed, ranging by then from 77 to 100% of the total amount excreted. It is concluded that lactulose in man is absorbed and excreted only in very small amounts and is not metabolized.

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EFFECTS OF PROLONGED INGESTION OF XYLOSE ON RATS

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(Received for publication August 20, 1952)

Xylose is a pentose sugar which might conceivably be incorporated into foods for human consumption. In terms of sweetness it has a value of 40 when compared to sucrose at 100. Monogastric animals are unable to utilize this 5-carbon sugar (Pflüger, '05; Miller and Lewis, '32; Anderson, '50), which suggests that it might be used as a dietary sweetening agent by individuals who desire to reduce body weight. Xylose might also serve as a sweetening agent for diabetics, either in addition to or in lieu of saccharin. The tendency of this sugar to cause diarrhea in laboratory animals suggests its possible use in laxative preparations. In the event a use is found for xylose, it could be produced in quantity from cottonseed hulls and corn cobs, thanks to the methods of production developed by Dunning and Lathrop ('45).

The possible inclusion of xylose in foods immediately raises the question as to whether any health hazard would be involved. The literature concerning the utilization of this substance by man is neither very extensive nor conclusive, but some experimental work has been done using laboratory animals. Darby and Day ('39) reported on the cataractogenic action of xylose in rats. These same workers (Darby and Day, '40) and others (Anderson, '50; Blatherwick et al., '36) observed diarrhea and abdominal distension when rats received this sugar orally. Blatherwick and co-workers ('36) fed

rats a diet containing 5% xylose for a period of 64 days. In addition to the diarrhea, they found albuminous degeneration of the liver cells, but these effects were not permanent.

The performance of rats on various levels of intake and on different basal rations for long periods of time has not as yet been adequately studied. The present work was undertaken to get some indication of how much xylose rats can ingest continuously without encountering any adverse effects.

EXPERIMENTAL

The xylose¹ was fed to rats as a supplement to three different basal diets. One was identical to, or a modification of, that used by Darby and Day ('39) and consisted (in percentage) of crude casein 18, salts 3, cod liver oil 2, butterfat 6, yeast 10, cornstarch 26, and monosaccharide 35. The monosaccharide consisted of xylose adjusted with glucose to total 35. A commercial laboratory ration² was also used, the added xylose being substituted at the expense of the entire ration. A third basic diet was primarily a mixture of natural feeds, and is the diet most commonly used in this laboratory. It is described in a previous report (Wilson and DeEds, '50) as the Addis diet. Obviously these latter two feed mixtures would be somewhat comparable to human diets.

Unless otherwise indicated, the rats were weanling Sprague-Dawley females. They were housed in individual wire-bottom cages and received tap water and the particular diet ad libitum. A weekly record of body weights and food consumption was maintained. Five rats were used per group.

Ophthalmoscopic examinations of the eyes were made during the entire experimental period at weekly intervals. Before examination the iris was dilated by instillation of a drop of 0.5% atropine sulfate solution into each eye.

¹Obtained from the Northern Regional Research Laboratory, Peoria, Illinois. It gave the following analysis: $[\alpha]_D^{25} = +19.5^\circ$; ash 0.20 to 0.37%.

²Purina laboratory chow. (Mention of this product does not imply that it is endorsed or recommended by the Department of Agriculture over others of a similar nature not mentioned.)

All rats surviving the 20-week feeding period were autopsied and examined grossly, and tissues were saved for histological study.

Diabetic rats were obtained by injecting alloxan. Various dosage levels were tried as well as subcutaneous, intraperitoneal, and intra-cardiac routes of administration. The criterion for selection of diabetic rats was the presence of elevated blood sugar levels, determined by the method of Folin and Malmros ('29). Polyuria was another evidence of diabetes. The blood sugar levels were used as a basis for dividing the diabetic rats into two comparable groups, one being fed the Addis basal and the other receiving the Addis basal plus 15% xylose ad libitum. By so doing, the influence of the level of blood sugar on the extent of the lens opacity was equalized.

RESULTS

Using albino rats from our own colony (Wilson, DeEds and Cox, '41), it was observed that 21-day-old rats developed severe lens opacities in less than 10 days after receiving an Addis or Darby and Day basal ration containing 35% xylose. Comparable rats placed on these same diets, but at 35 to 40 days of age, had failed to develop any lens opacities after 5 weeks. Mitchell ('36) encountered similar results when feeding rats lactose or galactose, and demonstrated that age and strain have a marked effect on the susceptibility to cataracts.

In table 1 the results of two separate experiments involving the prolonged feeding of xylose to weanling rats have been consolidated. In the last column, titled "Ophthalmoscopic observation," it can be seen that no lens opacities were noted until the level of xylose constituted at least 15% of the diet. This appeared to be the critical level, regardless of which of the three basic diets was used. As the level was raised above 15%, there was a proportionate increase in the severity of the opacities observed. An arbitrary scale was established to indicate the degree of opacity as slight, moderate, severe or complete, depending on whether the area of opacity rep-

resented one-fourth, one-half, three-fourths or the entire lens area. Without exception the opacity was first observed in the periphery of the lens as a diffuse cloudiness, which then became more dense and progressed toward the center of the lens. When the intake of xylose was more than 20% of the diet, total lens opacity (no reflected light when the lens was examined with the ophthalmoscope) invariably resulted within three weeks.

A regression of lens opacity (recovery of lens transparency) was an unexpected observation in all but two rats. This regression was usually well underway within 60 days from the time of development of maximum opacity, but it should be emphasized that the regression was never complete. Thin circular vestigial lines of opacity, with transparent lens tissue both within and without the circle, were still visible with the ophthalmoscope even after 140 days, whether the rats were continuously receiving xylose in the diet or were taken off the experimental diet after only 21 days. Whether these thin dark circular lines were detrimental to normal vision is not known.³

Without exception diarrhea was noted within 48 hours after feeding this sugar at levels of 15% or more, but following a few weeks of adjustment the abnormal softness of the feces became less noticeable. The observation of bloat was suggestive of an abnormal fermentation in the digestive tract, due to xylose ingestion, but it was confined to the 25 and 35% groups and not all rats in a given group were affected.

From an inspection of table 1 concerning food intake and growth rates, it can be seen that group 13 (receiving 25% xylose plus the Addis diet) averaged only 207.6 gm in body weight, compared to group 2 (control) which weighed 254.4 gm after 20 weeks. This depression in growth was found to be highly significant when the individual body weights of the

³In the publication by Fitzhugh and Buschke ('49) there is a picture (fig. 3) of a rat lens (from an animal which had been fed β -tetralol) containing a dark circular line of opacity which strikingly resembled opacities which had regressed in our xylose-fed rats.

TABLE 1
Effect of xylose on food consumption, body weight, and lens transparency

DIET	DAYS ON EXPT.	AVERAGE FOOD CONSUMPTION GM/RAT/DAY	FINAL AVERAGE BODY WEIGHT	OPHTHALMOSCOPIC OBSERVATION
1. Darby and Day basal (DD)	140	10.6	213.4	No lens opacity.
2. Addis basal	140	13.5	254.4	No lens opacity.
3. DD + 5% xylose	140	10.8	223.8	No lens opacity.
4. Addis + 5% xylose	140	12.5	246.8	No lens opacity.
5. Purina + 5% xylose	140	15.3	226.6	No lens opacity.
6. Addis + 10% xylose	140	13.8	226.4	No lens opacity.
7. DD + 15% xylose	140	11.4	211.2	3 of 5 rats, moderate lens opacities, then regression.
8. Addis + 15% xylose	140	12.6	224.2	2 of 5 rats, slight lens opacities, then regression.
9. Addis + 15% xylose	140	13.1	210.4	2 of 5 rats, moderate lens opacities, then regression.
10. Purina + 15% xylose	140	16.2	223.2	3 of 5 rats, slight lens opacities, then regression.
11. Addis + 20% xylose	140	13.9	212.7	5 of 5 rats, severe lens opacities, then regression.
12. DD + 25% xylose	21	5 of 5 rats, complete lens opacities, then regression.
13. Addis + 25% xylose	140	13.4	207.6	10 of 10 rats, complete lens opacities, 8 of 10 regression.
14. Purina + 25% xylose	21	5 of 5 rats, complete lens opacities, then regression.
15. DD + 35% xylose	21	4 of 4 rats, complete lens opacities, then regression.
16. Addis + 35% xylose	21	5 of 5 rats, complete lens opacities, then regression.

rats in these two groups were treated statistically. The food consumption for these two groups was almost the same; namely, 13.4 and 13.5 gm per rat per day, respectively. However, since added xylose is not metabolized for energy or growth, the actual or corrected net food intake would be 25% less than 13.4 gm, or 10.05. Conversely, in order for rats receiving 25% xylose to gain weight equal to that gained by the controls, the food intake would have to be $13.5/0.75$, or 18 gm. It appears, then, that the intake of utilizable food was reduced (voluntary semi-starvation) as the per cent of xylose in the ration increased. This observation is in agreement with that of Anderson ('50).

Concerning survival rates, only two rats failed to survive the 140-day experimental regimen, out of a total of 60 animals. Since there were no deaths on the highest level of xylose (25%), it is concluded that the ingestion of xylose did not increase mortality.

At the end of the experiment all surviving rats were killed by ether and decapitation. Post-mortem examination for gross pathological changes revealed no abnormalities other than lung infections scattered throughout all groups. Sections of the following tissues were studied by Dr. A. J. Cox, Jr., Department of Pathology, Stanford University School of Medicine, San Francisco, California: thyroid, heart, lungs, liver, spleen, kidney, adrenal, intestine, pancreas and ovary. The only change directly related to the amount of xylose in the diet was seen in the liver. Livers from animals which received 25 or 35% xylose for 37 to 39 days showed definite, though slight, swelling of the cytoplasm of the hepatic cells. There was some separation of the stained particles within the cytoplasm but no definite vacuolation was seen in most of the cells. The nuclei appeared normal and there was no reason to conclude that the liver cells had been injured. The appearance was suggestive of a storage phenomenon. Livers from rats which received 15% xylose for 22 weeks also showed some similar swelling of the cytoplasm. This was not definite in the animals receiving 5% xylose.

As mentioned earlier, xylose may be ingested by diabetics. Medical records substantiate the fact that the incidence of lens opacities in diabetics is appreciably higher than for non-diabetics (Adler, '48). Would super-imposing of one cataractogenic agent upon another enhance the formation of cataracts? Attempts were made to feed xylose to rats made diabetic through the use of alloxan, according to the procedure described by Kass and Waisbren ('45). The yield of alloxan-diabetic rats was so low that out of 223 treated animals of different ages, sex, and strain, only 19 survivors were obtained with blood sugar at a level above 200 mg per 100 ml, of which 7 died before cataracts developed. Six diabetic rats, maintained on the Addis ration only, developed cataracts in approximately 41 to 57 days. However, 6 diabetic rats fed the same basal ration containing 15% xylose experienced cataracts as soon as the controls, but with a progressively much greater loss of lens transparency with respect to time. In fact, when the lens opacities were visible to the naked eye in the rats receiving xylose, the opacities in the controls were not noticeable. Greater numbers of animals will be required to verify these results, but the severity of the cataracts was decidedly in the direction of the xylose-fed rats.

DISCUSSION

The question is raised as to why xylose-fed weanling rats initially got rather severe lens opacities and then with time regained much of the lost transparency. There is no apparent answer to this question. One can only point out that the observed spontaneous reversibility of the lens opacity was in accord with the finding that in older rats the incidence of cataracts is either absent or greatly diminished when xylose is fed. Bellows and Chinn ('41) speculated that the basic cause of lens opacities was a non-specific osmotic derangement, either local or systemic. Such a mechanism would explain why a fairly large list of chemically unrelated compounds have been found capable of producing cataracts. These

same workers also concluded that the regression of experimental cataractous changes in the early stages points to a physical alteration which is reversible, rather than to irreversible chemical changes.

The results of the present study confirm the rather limited observations reported in the literature that xylose is toxic. If we can assume that humans would react in the same manner as rats, then xylose should not be incorporated into foods. It is true that the effects of xylose on the liver cells were reversible (Blatherwick et al., '36) and that a relatively high level of 15% in the diet was required to get detectable lens opacities in rats. More work would be required to determine whether it would be safe at lower levels when the ingestion was continued for longer periods. Again assuming that rats and humans would react in a comparable manner, it would seem that xylose might be more toxic for individuals with a tendency towards cataract (diabetics, for example) than for normal individuals.

SUMMARY

Xylose was fed for periods up to 140 days to rats at various levels from zero to 25% of the diet. Thirty-five per cent of xylose was fed for short periods. In weanling rats, a level of 15% or more was found to produce lens opacities, which regressed to a considerable degree with time whether or not the animals were continued on the experimental diet. The regression was never complete. Other toxic symptoms observed were diarrhea, bloating, and liver changes suggestive of storage. A voluntary restriction of utilizable food by the xylose-fed rats led to decreased rates of growth. Rats rendered diabetic with alloxan experienced more severe lens opacities when fed xylose than did diabetic controls. Pending more favorable experimental data at lower levels of intake, it is deemed inadvisable to risk the incorporation of xylose in foods at any level of intake for extended periods of time.

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II. RELATIVE SWEETNESS

Perceived sweetness is subjective and depends on or can be modified by a number of factors. The chemical and physical composition of the medium in which the sweetener is dispersed has an impact on the taste and intensity. The concentration of the sweetener, the temperature at which the product is consumed, pH, other ingredients in the product, and the sensitivity of the taster are all important. Again, sucrose is the usual standard. Intensity of the sweetness of a given substance in relation to sucrose is made on a weight basis. Table 1 provides the approximate relative sweetness of many of the alternative sweeteners discussed in this book.

Table 1 Relative Sweetness of Alternatives to Sucrose

	Approximate sweetness (sucrose = 1)
Lactitol	0.4
Hydrogenated starch hydrolysates	0.4–0.9
Trehalose	0.45
Isomalt	0.45–0.65
Isomaltulose	0.48
Sorbitol	0.6
Erythritol	0.7
Mannitol	0.7
Maltitol	0.9
D-Tagatose	0.9
Xylitol	1.0
High fructose corn syrup, 55%	1.0
High fructose corn syrup, 90%	1.0
Crystalline fructose	1.2–1.7
Cyclamate	30
Glycyrrhizin	50–100
Aspartame	180
Acesulfame potassium	200
Saccharin	300
Stevioside	300
Sucralose	600
Hernandulcin	1000
Monellin	1500–2000
Neohesperidine dihydrochalcone	1800
Alitame	2000
Thaumatococin	2000–3000
Neotame	8000

Inulin and oligofructose do not influence the absorption of cholesterol, or the excretion of cholesterol, Ca, Mg, Zn, Fe, or bile acids but increases energy excretion in ileostomy subjects

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Objective: To investigate the effects of inulin and oligofructose on cholesterol absorption and excretion of cholesterol, bile acids, energy, nitrogen and minerals in man.

Design: Double-blind cross-over study.

Setting: Metabolic kitchen with polyclinic visits, Sahlgrenska Hospital, Göteborg, Sweden

Subjects: Patients with conventional ileostomy because of ulcerative colitis.

Interventions: 17 g of inulin, 17 g of oligofructose and 7 g of sucrose were added to a controlled diet during three experimental periods of three days each. Ileostomy effluents were collected and analysed. Differences between experimental and control diet were investigated with the Wilcoxon's sign and values test.

Results: Inulin and oligofructose were recovered in the ileostomy effluent to 88% (95% CI, 76–100%) and 89% (64–114%) respectively. Dry solid excretion increased by 14.4 g (11.3–17.5) on inulin, and by 14.7 g (13.0–16.4 g) on oligofructose and energy excretion increased 245 kJ (190–307 kJ) on inulin and 230 kJ (217–315 kJ) on oligofructose compared to control diet ($P < 0.05$). Cholesterol absorption, excretion of cholesterol, bile acids, nitrogen, fat, calcium, magnesium, zinc and iron were not affected by inulin and oligofructose.

Conclusions: Inulin and oligofructose are not digested in the small intestine. They do not affect mineral excretion and hence hardly mineral absorption. They do not increase fat or nitrogen excretion from the small intestine. Any physiological effect of inulin and oligofructose is probably mediated through other mechanisms than altered excretion from the small intestine.

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Descriptors: inulin, oligofructose, ileostomy, cholesterol, absorption, minerals.

Introduction

Dietary fibre is a general term covering substances which resist hydrolysis in the stomach and the small intestine (Trowell, 1974), and hence escape digestion in the small intestine. Inulin from chicory is a set of $\beta(2-1)$ fructans with a chain length between 2 and 60 or more. Oligofructose is a subset of inulin obtained by enzymatic hydrolysis of inulin, with a degree of polymerisation (DP) varying between 2 and 8. Inulin and oligofructose are present as such in many plants where they provide a considerable part of the stored energy (Van Loo *et al*, 1995). Wheat flour contains 1–4% fructans on solid matter, artichoke 20–65%, asparagus 30%, onion up to 50%. They are apparently not digested in the small intestine but are readily fermented in the large intestine, and could thus be classified as dietary fibre (Roberfroid, 1993).

In a typical European diet, less than 20 g of dietary fibre are consumed daily (Cummings, 1995). During the last decades much attention has been paid to the physiological effects of different dietary fibres. Non-fermentable dietary fibres, such as wheat-bran, reduce transit time but hardly affect lipid metabolism, whereas fermentable fibres such as pectins and β -glucans, reduce serum cholesterol but hardly affect transit time (Truswell, 1995).

It is reasonable to assume that the main effects of high-fibre diets on sterol metabolism is due to mechanisms in the small bowel (Truswell, 1995), although an effect on cholesterol synthesis by propionate from increased fermentation in the colon has been proposed from animal experiments. (Chen *et al*, 1984). A close correspondence has been reported between the excretion of cholesterol and bile acids from the small bowel and the effects on serum cholesterol levels (Andersson and Bosaeus, 1993).

Much less attention has been paid to the physiological effects of inulin and oligofructose, although at least a few grams of inulin and oligofructose are consumed daily (Van Loo *et al*, 1995). Some animal experiments and observations in humans suggest a serum-cholesterol-lowering effect of inulin and oligofructose (Fiordaliso *et al*, 1995; Davidson *et al*, 1996).

Preliminary reports have shown beneficial effects of fructooligosaccharides on diabetic control and serum cholesterol levels (Yamashita *et al*, 1984), but the definitive action is still unclear. Patients with conventional ileostomy after total colectomy offer a possibility to investigate the quantity of dietary fibre, minerals, and nutrients, which in normal subjects pass into the large intestine (Sandberg *et al*, 1981). Moreover, small bowel excretion of cholesterol and bile acids as well as cholesterol absorption by isotopic techniques, could be studied at the same time (Andersson, 1992).

Table 1 Clinical data on ileostomy subjects

Subject number	Sex	Age	Body weight	Serum cholesterol	Serum triglycerides	Medication
		years	kg	mmol/l	mmol/l	
1	F	30	57	4.5	0.8	
2	M	41	75	4.3	1.0	
3	M	54	87	5.0	1.0	Thyroxin
4	M	67	77	8.2	1.8	—
5	F	6-	62	6.8	1.1	—
6	M	41	60	7.9	0.9	—
7	F	41	62	4.7	0.7	Sumatriptan
8	F	69	52	7.3	1.5	—
9	F	67	69	8.1	0.7	—
10	M	71	79	6.4	1.2	Prednisolon
Mean		54	68	6.3	1.1	

This study was performed to investigate the digestibility in man of inulin (Raftiline® ST) and oligofructose (Raftilose P95) using ileostomy subjects. In addition the study was designed to investigate the effects of inulin and oligofructose on the absorption of cholesterol, and the excretion of cholesterol, bile acids, calcium, magnesium, zinc, iron, and energy from the small bowel.

Study design and subjects

Study protocol

The study was designed as a double-blind cross-over trial with 10 subjects acting as their own controls. Diets with inulin or sucrose to identical taste were given for 3 d each in random order. In addition and identical diet with oligofructose was given to 8 of the initial 10 subjects after the first two diets. These 8 subjects were studied during three 3 d periods, on the same weekdays on each diet during three consecutive weeks. The first day of each period was used for adaptation and to minimize any carry-over effects from the habitual diets of the participants. Between the dietary periods there was a wash-out period of 4 d. Informed consent was obtained from the study participants. The study protocol was approved by the ethical committee of Sahlgrenska Hospital.

Subjects

Clinical data on subjects are given in Table 1. Ten subjects (5 women and 5 men) with conventional ileostomy due to ulcerative colitis volunteered for the study. Mean age was 54 y (range 30–71 y) and mean body weight 68 kg (range 52–87). No additional intestinal resection had been performed, and all had stable ileostomy functions. No signs of additional thyroid, renal, pancreatic, hepatic disease, inflammation or anaemia were revealed through medical history or standard laboratory tests. The mean serum cholesterol level was 6.3 (s.d. 1.6) mmol/l and mean serum triglyceride level 1.1 (s.d. 0.4) mmol/l.

Materials and methods

Food

All food was prepared in advance in a metabolic ward kitchen from the same batches of fat, meat, fish and flour for the inulin and sucrose periods. All items were weighed to nearest 0.1 g on a Sigma scale. For the oligofructose periods the fat batches were identical with the earlier balance periods. Dishes were stored deep-frozen in special containers until served in the same containers to minimise losses. Composition of the control diet is given in Table 2.

19.4 g of inulin was given during the inulin period; (Raftiline®ST), containing 92% inulin on dry solids and a DS content of 95%, with an average degree of polymerisation of 10 (range 2–65) corresponding to a theoretical amount of 17.0 g of pure inulin. During the oligofructose period 19.0 g oligofructose was given; (Raftilose®P95), containing 95% oligofructose on dry solids and a DS content of 95%, with an average degree of polymerisation of 4 (range 2–8) corresponding to a theoretical amount of 17.1 g of pure oligofructose. Inulin and oligofructose were added to the recipes and included in the dishes, divided into three doses given at breakfast, lunch, and dinner. In the sucrose period 7.5 g of sucrose was added to the recipes in order to make the dishes indistinguishable.

On the second day 4.6 kBq (125 nCi) of [β -4-C14]-sitosterol (specific activity 20 kBq/mmol), and 19.2 kBq (250 nCi) of [1 α , 2 α -H3]-cholesterol (specific activity 1.79 MBq/mmol) were added in three equal doses to breakfast, lunch and dinner. Isotopes were purchased from Amersham International, Buckinghamshire, UK.

Breakfast consisted of cheese rolls, orange juice, coffee or tea. Cod with broccoli and sauce béchamel was served with polished rice for lunch. Dinner was composed of filet of pork with mushroom sauce and boiled potatoes together with lettuce and a banana for desert. Tea, coffee and rolls were served as snacks and the evening meal. Breakfast was served in the metabolic kitchen at the department, and food for the rest of the day was given to each subject in the morning.

No other food was allowed during the study period. Drinks were coffee, tea, mineral water or light beer, in fixed amounts for each individual during all study periods. The diet was composed of ordinary food items with the same menu in all study periods. All meals were eaten at fixed times. To meet the different energy requirements for each subject special rolls were prepared. The rolls had the same proportions of fat, protein, carbohydrate, cholesterol and fibre as in the basal menu. The composition of the diet was calculated from extended food composition tables (Swedish Food Administration, 1992) using a software PC-Dietist 5.6 (Kost Näringsdata, Stockholm, Sweden), and from analyses of energy, nitrogen, minerals, cholesterol and dietary fibre.

Analyses

Ileostomy effluents were collected on the second and third day of each dietary period in seven plastic bags every second hour during the day (8 am to 10 pm), once or twice during the night and immediately frozen on dry ice in thermos vessels (Karsruher glastechnik, Karlsruhe, Ger-

Table 2 Composition of the control diet to which inulin or oligofructose was added in exchange for sucrose

Basal diet		
Energy ^a	MJ	10.0 ± 0.2
Nitrogen ^a	g	14.2 ± 0.06
Protein (N × 6.25)	g	89 ± 0.4
Fat	g	99
E% Protein		15
E% Fat		40
E% Carbohydrates		45
E% Saturated Fat		20
E% Polyunsaturated fat		5
Cholesterol ^a	mg	288 ± 7
Plant sterols ^a	mg	283 ± 3
Calcium ^a	mg	1130 ± 6
Magnesium ^a	mg	304 ± 15
Total dietary fibre ^a	g	19 ± 1.6
Fructooligosaccharides ^a	g	< 1 (in inulin diet 17.1, in oligofructose diet 15.5)

^a = analysed values.

Remaining values calculated from food tables (Swedish Food Administration 1992).

Table 3 Excretion of effluent and nutrients, and absorption of cholesterol in 10 patients with conventional ileostomy on control diet + 8 g sucrose, control + 16 g oligofructose

Excretion		Sucrose (n = 10)	inulin (n = 10)	Oligofructose (n = 8)
Wet weight	g	793 (582–1005)	822 (646–999)	739 (583–894)
Dry weight	g	62.0 (52.4–71.6) ^a	76.5 (67.8–85.1)	74.0 (67.9–80.2)
Nitrogen	g	2.2 (1.9–2.5)	2.3 (2.0–2.7)	2.2 (2.0–2.4)
Fat	mmol	8.7 (6.3–11.2)	9.6 (7.4–11.7)	9.3 (7.4–11.2)
Energy	kJ	1030 (870–1180) ^a	1275 (1150–1400)	1260 (1150–1360)
Cholesterol	mg	506 (392–620)	504 (382–626)	501 (386–616)
Bile acids	mg	480 (265–696)	453 (259–698)	326 (192–459)
Calcium	mmol	29.3 (23.3–35.2)	29.8 (25.0–34.5)	31.2 (25.5–36.8)
Magnesium	mmol	11.1 (8.7–15.4)	10.9 (9.1–12.8)	10.8 (9.3–12.4)
Zinc	μmol	119 (100–138)	147 (98–198)	116 (100–132)
Iron	μmol	318 (262–373)	326 (252–393)	282 (252–313)
Starch	g	8.8 (6.7–10.8)	9.1 (7.1–11.0)	8.6 (6.5–10.8)
Fructooligosaccharides	g	< 1	15.0 (13.0–17.0)	13.8 (9.8–17.7)
Cholesterol absorption	%	63 (57–69)	60 (56–66)	62 (54–70)

^a = $P < 0.05$ as compared to the other diets.

many). The effluents were then lyophilised to constant weight and meticulously ground and mixed prior to analysis. Duplicate portions of the diets were mixed and lyophilised in the same way. Energy content was measured by combustion in a Gallenkamp (Loughborough, Leicestershire, UK) bomb calorimeter.

Fat in ileostomy effluent was determined according to van de Kamer (van de Kamer *et al.*, 1949). Nitrogen was assayed by a modified micro-Kjeldahl technique (Sandberg *et al.*, 1981). Magnesium, calcium, iron and zinc were determined on a atomic absorption spectrophotometer. Dietary fibre except fructans was measured by the AOAC-method after enzymatic hydrolysis according to Asp (Asp *et al.*, 1983). Cholesterol, plant sterols and (in ileostomy effluents) bile acids were determined by gas liquid chromatography as described previously, with a coefficient of variation of 2.6–4% (Bosaeus and Andersson, 1987). Isotope activities were assayed by liquid scintillation counting in a Beckman Tricarb 1900TR as described previously (Ellegård and Bosaeus, 1994). Variation in duplicate analysis were 3.3% for [3H]-cholesterol and 2.8% for [14C]-β-sitosterol.

Inulin and oligofructose were determined as fructose and glucose by capillary gas chromatography before and after complete enzymatic hydrolysis (Quemener *et al.*, 1994; De Leenheer and Hoebeys, 1994; Van Loo, 1996).

Calculations and statistical methods

Values on wet weight, dry weight, bile acid, fat, nitrogen, calcium, magnesium, iron, zinc and energy excretion are reported as means for each dietary period. Net cholesterol and net sterol excretion were calculated as follows

$$\begin{aligned}\text{Net cholesterol excretion} &= \\ &\text{cholesterol in effluent} - \text{cholesterol in food} \\ \text{Net sterol excretion} &= \\ &\text{bile acids in effluent} + \text{net cholesterol excretion}\end{aligned}$$

Fractional cholesterol absorption was calculated from isotope-ratios for the first 24 h following isotope ingestion using the equation of Quintao (Quintao *et al.*, 1971).

$$(1 - (\text{radioactivity in faecal H-3-cholesterol} / \text{radioactivity in faecal C-14-beta-sitosterol}) \times (\text{radioactivity in administered C-14-beta-sitosterol} / \text{radioactivity in administered H-3 cholesterol})) \times 100.$$

Absolute absorption of cholesterol was calculated as: (cholesterol intake × fractional cholesterol absorption).

All descriptive values are given as mean for 24 h with 95% confidence intervals unless otherwise stated. Comparisons of data for the dietary periods were made by Wilcoxon's signed ranks test, using Systat 5.1, (Systat Inc. Evanston, Ill, USA) for the calculations.

Results

None of the subjects noticed any difference between the three diets, indicating that doses of 17 g of inulin and oligofructose are well tolerated. The fructan diets resulted in higher ileostomy dry weights compared to the sucrose diet, although there was no significant increase in total ileostomy output as measured by wet weight. The amount of inulin recovered from the inulin diet was 17.1 g according to analysis and the amount of oligofructose recovered from the oligofructose diet was 15.5 g, compared to the theoretical figures of 17.0 g and 17.1 g respectively. Data on excretion and absorption are given in Table 3.

There was 14.4 g and 14.7 g more excretion calculated as dry weight from the inulin and oligofructose diets respectively. The excretion of fat and nitrogen was hardly affected by diet, although there was a tendency to higher excretion in the fructan periods. Energy excretion was 245 kJ (190–307) and 230 kJ (217–315) higher on the inulin and oligofructose diets as compared to the sucrose diet. Mean excretion of inulin in ileostomy effluent was 15.0 g (13.0–17.0) corresponding to 88% (76–100%) inulin recovery whereas mean oligofructose excretion was 13.8 g (9.8–17.7) corresponding to 89% (54–114%) oligofructose recovery.

Total starch was excreted to the same amount on all three diets.

The excretion of minerals was also totally unaffected by inulin and oligofructose. Excretion of cholesterol and bile acids showed no difference between the diets. Cholesterol absorption averaged 63% (57–69) on the sucrose diet, and was practically unaffected by oligofructose and inulin. Mean recovery of added non-absorbable β -sitosterol was 89% within 24 h and 94% within 48 h.

Discussion

Balance studies in ileostomy subjects make it possible to accurately detect rapid changes in absorption and excretion over the small bowel, mainly due to short transit time and minimised bacterial degradation (Andersson, 1992a). In the present study 17.1 g of inulin, 15.5 g of oligofructose and 7.5 g of sucrose were given to ileostomy subjects on a controlled western-style diet. The fructans were well tolerated, and were recovered in the ileostomy bags by 88% for inulin and 89% for oligofructose. These recoveries are similar to the recovery of the non-absorbable steroid sitosterol, and are in close agreement with other studies on inulin excretion in ileostomy subjects. (Bach-Knudsen and Hesso, 1995). Identical recoveries of arabinose (88%), xylose (89%) and glucose (89%) from wheat bran have been reported from an original ileostomy study from our department (Sandberg *et al.*, 1981). Thus, inulin and oligofructose in the preparations used in this study are resistant to digestion in the human stomach and small intestine. The inulin was unaffected by the preparation of the diet whereas the actual amount of oligofructose was lower than theoretically calculated, 15.5 and 17.7 g respectively, indicating a possible 10% hydrolysis during preparation.

The fructans do not seem to affect the apparent absorption of nutrients from the small bowel as the excretion of nitrogen, fat, calcium, magnesium, zinc, iron, cholesterol and bile acids were virtually the same on all three diets. The current AOAC-method for determination of dietary fibre, as used in this study, does not include fructans.

Earlier studies in ileostomy subjects have shown a

correlation between the serum cholesterol lowering property of a dietary fibre product and small bowel excretion (Anderson, 1992). Citrus pectin (Bosaeus *et al.*, 1986) and oat bran (Zhang *et al.*, 1992), known to reduce serum cholesterol increased ileal bile acid excretion. Wheat bran, without effect on serum lipids, did not induce any significant change in sterol excretion (Bosaeus *et al.*, 1986). Ileostomy studies with 3 days on each dietary period, with the same number of subjects, given 15 g pectin, or 15 g of mixed dietary fibres from unrefined food, could detect changes in sterol excretion of 15%. (Andersson and Bosaeus, 1993). Thus, from this study, it seems unlikely that inulin and oligofructose in the similar doses would induce such effects on sterol (or lipid) excretion from the small bowel.

However, an extraintestinal hypocholesterolaemic mechanism has been advocated. Propionate, after absorption from the colon as a fermentable product from carbohydrate breakdown, has been suggested to suppress cholesterol synthesis in rat liver (Chen *et al.*, 1984). Different experiments in animals to test this hypothesis have given conflicting results (Nishimura *et al.*, 1993; Moundras *et al.*, 1994; Fiordaliso *et al.*, 1995). In man (Lairon, 1996), however, it seems unlikely that this mechanism may be of clinical importance as oat bran reduces LDL-cholesterol levels considerably in ileostomy subjects (Zhang *et al.*, 1991), who only produce insignificant amounts of propionate (Cummings and Englyst, 1991). Moreover, in vitro experiments have shown human hepatocytes to be much less sensitive to propionate than rat hepatocytes (Lin *et al.*, 1995), and the different results could thus be due to species differences.

Inulin and oligofructose have profound effects on the colonic microflora. The endogenous Bifidobacteria are favoured by these substances, whereas other strains of bacteria are constricted. (Gibson *et al.*, 1995). The present study shows that almost 90% of the inulin and oligofructose in the diet pass through the small bowel and that the absorption of other nutrients seems to be unaffected by these substances. The energy contribution to the colon increases about 25% by the intake of 16–17 g of inulin and oligofructose to the basal experimental diet. The increase in energy excretion (246 and 230 kJ) respectively is neatly accounted for by the excretion of 14.5 g inulin and 13.8 g oligofructose which yield 246–235 kJ as measured by bomb calorimetry. This increase should be sufficient to change the dominant bacterial pattern in faecal analyses (Gibson *et al.*, 1995).

Inulin and oligofructose are recovered to approximately 90% from ileostomy effluents, suggesting only minor hydrolysis or bacterial degradation during intestinal passage. The latter seems unlikely as there are only traces of secondary bile acids, as a sign of bacterial degradation, present in the ileostomy effluents. However, some degradation of inulin and oligofructose in the ileostomy bags cannot be excluded. Bach-Knudsen and Hesso (1995) found increased amounts of short chain fatty acids in a similar ileostomy study during less frequent sample collection during the night, compared to every 2 h during daytime. This indicates a possible post-ileum polysaccharide degradation, but it seems to amount to 10% degradation of inulin and oligofructose at the most.

In conclusion, the present study did not show any effects of 16–17 g of inulin or oligofructose on the absorption of cholesterol, or the excretion of fat, cholesterol, bile acids, calcium, magnesium, zinc or iron. Thus, the systemic

effects of inulin and oligofructose reported previously are not mediated via increases in cholesterol and sterol excretion, proposed to explain the hypocholesterolemic effect of other soluble dietary fibres such as pectins, gums, and β -glucans.

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Fruit Juice Consumption by Infants and Children: A Review

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Key words: child nutrition, fruit juice, beverages, diet, nutrition policy, growth disorders

The pattern of fruit juice consumption has changed over time. Fifty years ago, orange juice was the major juice produced and it was consumed primarily to prevent scurvy. Now, apple juice is the juice of choice for the under 5 age group. While fruit juice is a healthy, low-fat, nutritious beverage, there have been some health concerns regarding juice consumption. Nursing bottle caries have long been recognized as a consequence of feeding juice in bottles, using the bottle as a pacifier, and prolonged bottle feeding. Non-specific chronic diarrhea or "toddler's" diarrhea has been associated with juice consumption, especially juices high in sorbitol and those with a high fructose to glucose ratio. This relates to carbohydrate malabsorption, which varies by the type, concentration, and mixture of sugars present in different fruit juices. Fruit juice consumption by preschoolers has recently increased from 3.2 to about 5.5 fl oz/day. Consumption of fruit juice helps fulfill the recommendation to eat more fruits and vegetables, with fruit juice accounting for 50% of all fruit servings consumed by children, aged 2 through 18 years, and 1/3 of all fruits and vegetables consumed by preschoolers. Concomitant with the increase in fruit juice consumption has been a decline in milk intake. This is concerning as milk is the major source of calcium in the diet, and at present, only 50% of children, aged 1 through 5 years, meet the RDA for calcium. Studies of newborn infants and preschool-aged children have demonstrated a preference for sweet-tasting foods and beverages. Thus, it is not surprising that some children, if given the opportunity, might consume more fruit juice than is considered optimal. Eleven percent of healthy preschoolers consumed ≥ 12 fl oz/day of fruit juice, which is considered excessive. Excess fruit juice consumption has been reported as a contributing factor in some children with nonorganic failure to thrive and in some children with decreased stature. In other children, excessive fruit juice consumption has been associated with an increased caloric intake and obesity. This paper reviews the role of fruit juice in the diets of infants and children and outlines areas for future research. Recommendations regarding fruit juice consumption based on current data are also given.

Key teaching points:

- Fruit juice consumption by infants and children has increased in recent decades.
- Fruit juice accounts for 50% of all fruit servings consumed by children.
- Fruit juice accounts for one-third of all fruit and vegetable servings consumed by preschoolers.
- Excess fruit juice consumption is associated with failure to thrive and short stature in some children.
- In other children, intake of excess fruit juice is associated with obesity.

Commercial production of orange juice began some 50 years ago following a surplus of oranges. Since then, there has been an increase in the varieties of fruit juice produced as well as an increase in the marketing and distribution of fruit juices. This has been accompanied by an increase in the consumption of fruit juices by infants and children. Fruit juice is a good tasting, convenient snack food that is favorably priced com-

pared to milk or soda pop. It is also perceived and promoted as a healthy, non-fat, often natural, nutritious drink. Recent national public health programs [1,2] and manufacturers' marketing efforts designed to promote increased consumption of fruits and vegetables also may have contributed to recent increases in fruit juice consumption.

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FRUIT JUICE IN INFANTS' DIETS

During the first 4 to 6 months of life healthy infants fed only breast milk or infant formula require little or no water and no fruit juice [3]. When solid foods are introduced, additional water is often required because the renal osmolar load is high in foods with a higher protein or electrolyte content such as strained meat, egg yolk, and high meat dinners. While fruit juices, fruits, puddings, vegetables, and desserts have a low renal solute load, it is recommended that infants be offered water, rather than fruit juice, as part of a feeding [3]. This allows them an opportunity to fulfill fluid needs without an obligatory intake of extra calories. There has been concern that some infants who cry because they are thirsty may be offered breast milk, formula, or fruit juice in the mistaken belief that crying indicates hunger [3].

During the 1940's and 1950's, Virginia Beal's classic dietary studies of infants and children documented the progressively earlier introduction of solid foods (cereal, fruits, vegetables, meats, and "meat soup") by parents and caretakers [4]. She attributed this, in part, to the easier availability of pureed and specially prepared foods for infants. In addition, the "mother's desires to see their infants gain weight rapidly" and the "mistaken assumption that added solid foods helped the infant sleep through the night" may have also contributed to the earlier introduction of solid foods [5]. Beal noted, however, that even though infants were offered supplemental foods at earlier and earlier ages, the average age of infant acceptance of these solid foods changed little [4]. Most infants would accept cereal and fruit around 3–4 months, vegetables around 4–5 months and meats around 6 months. The only fruit juice discussed by Beal was orange juice which was then given to prevent scurvy. She notes that between 1946 and 1957 the average age of introduction of orange juice increased from 3 to 8 months, with an increasing dependence on the vitamin preparations as a source of ascorbic acid. During this same period, the age of transition from prepared baby foods to the family's diet decreased by almost half from approximately 2 years to 13 months.

In 1958 the American Academy of Pediatrics (AAP) Committee on Nutrition issued a statement recommending that solid foods not be introduced until 4 to 6 months of age [5], at which time breast milk alone may no longer be sufficient to provide the total nutrient needs of the baby [6]. "By 4 to 5 months of age, the extrusion reflex of early infancy has disappeared, and the ability to swallow nonliquid foods has become established. By 5 to 6 months of age, the infant will be able to indicate a desire for food by opening his or her mouth and leaning forward, and to indicate disinterest or satiety by leaning back and turning away. Until the infant loses the extrusion reflex, supplements of solid foods are difficult to give by spoon" [3]. In 1980, the Committee reiterated their statement because of the "continuing widespread and possibly harmful effects of introducing supplemental foods at 1 or 2 months of age or

earlier" [7]. The Committee also stated that "juices should be introduced when the child can drink from a cup" [3]. Juices provide carbohydrate, vitamin C, and water. By the 1990's, 90% of all infants, by 1 year of age, were consuming fruit juice [8], and baby juice sold in bottles was a \$164 million industry [9].

FRUIT JUICE CONSUMPTION

Children's Intake of Fruit Juices

According to juice manufacturers, infants consume, on average, 5 fl oz/day of fruit juice and children under 5 years of age consume 9 gallons per year (3.2 fl oz/day) [8]. In three recent studies the amount of fruit juice consumed by young children was higher, indicating a temporal change. In a recent study of low-income 4- and 5-year old Latino children, the children consumed a mean of 6.0 fl oz/day of fruit juice, with a median intake of 5.4 fl oz/day (based on up to 7 days of diet records collected between 1986–1989) [10]. In a recent study of rural, predominantly white, low- to middle-income children, the 2- and 5-year old children consumed a mean of 5.9 and 5.0 fl oz/day of fruit juice, respectively, based on 7 days of consecutive dietary records collected between 1992–1993 [11]. These children's median juice intake was 4.7 fl oz/day. In a study of 2- to 18-year old children (based on 3 days of dietary data collected by the US Department of Agriculture as part of the 1989–1991 Continuing Survey of Food Intakes by Individuals [CSFII]) the children's mean fruit juice consumption was 4.0 fl oz/day [12].

The definition of what constitutes a fruit or vegetable serving has been clarified recently by the US Department of Agriculture in their publication of The Food Guide Pyramid [13]. One serving of fruit is provided by 6 fl oz of fruit juice. Using these definitions, recent studies have found that fruit juice contributed substantially to the total number of servings of fruits eaten by children. In the study of young Latino children, fruit juices constituted 56% of all fruit servings consumed and 36% of all fruit and vegetables consumed (fried vegetables were excluded) [10]. These children had a mean intake of 1.8 servings/day of fruits and 1.0 servings/day of vegetables. In the study of young rural children, fruit juice contributed 53% of all fruit servings consumed and 35% of all fruits and vegetables consumed (excluding fried vegetables) [14]. These children had a mean intake of 1.6 servings/day of fruits and 0.7 servings/day of vegetables. In the study of 2- to 18-year old children in the CSFII, fruit juices accounted for 47% of all fruit servings consumed (unsweetened fruit juices contributed 41.5% and sweetened fruit juices contributed 5.2% of fruit servings) [12]. The male and female children, aged 2–5 years, had mean intakes of 1.5 and 1.6 servings/day of fruits and 1.8 and 2.0 servings/day of vegetables, of which 1.0 and 1.1 servings/day, respectively, were "starchy vegetables" including fried

vegetables. Fried vegetables (67% of which were french fries) comprised 34.5% of all vegetables consumed. Among the nutrition community there is debate about whether fried vegetables and/or "starchy vegetables" should be counted as vegetables. In the CSFII, children's intakes of fruits and vegetables increased slightly with rising income, while starchy vegetables showed the opposite trend [12]. In the study of rural children, consumption of fruit juices, fruits, and/or vegetables did not differ by primary caretaker education, by family participation in the food stamp program, or by participation in the Special Supplemental Food Program for Women, Infants and Children [11,14].

Fruit Juice Versus Milk Consumption

It has been suggested that fruit juice may be replacing milk in the diets of young children [9]. In the study of rural children, the 7-day median milk intakes for the 2- and 5-year old children were 8.8 and 9.1 fl oz/day, respectively [11], which is considerably less than the median milk intake of 16 fl oz/day reported for 2- to 3-year old children in 1957 [4]. The 2- and 5-year old children's mean milk consumptions were 9.8 and 11.0 fl oz/day, respectively, which is only slightly higher than the 9.1 fl oz/day reported to be consumed by US children and adults in 1992 [15]. Dairy products are the major source of calcium in the US food supply, currently supplying 75% of the dietary calcium in the US [16]. There has been a decrease in the level of calcium available in the US food supply since 1945-1949, when the use of dairy products was highest and a mean level of 1000 mg per capita per day was available, and the 1980's, when the mean levels available were 840-900 mg per capita per day [16]. Fluid milk provides about two-thirds of the calcium provided by dairy products. In the National Health and Nutrition Examination Survey (NHANES) III (1988-1991) the mean calcium intakes for 1- to 2-year old children and 3- to 5-year old children were 835 and 855 mg/day, respectively, with median intakes of 800 and 798 mg/day, respectively [17]. The optimal calcium requirement for children, aged 1 through 5 years, is 800 mg/day [18]. Therefore, only 50% of young children are meeting the recommended daily allowance (RDA) for calcium (i.e. have a calcium intake \geq 800 mg/day) [19]. While the decrease in milk and calcium intake parallels the increase in fruit juice consumption, consumption of soda pop has also increased during this same time period [16]. There are no data available to definitively link one with the other or to say that one is causative of the other. In a cross-sectional study of young children, there was no evidence that children drinking excessive amounts of fruit juice were substituting fruit juice for milk; children drinking \geq 12 fl oz/day of fruit juice were drinking the same amount of milk as children drinking less juice: 11.1 vs 10.2 fl oz/day of milk, respectively [11]. Current recommendations by nutrition experts to obtain optimal calcium intakes call for a "change in dietary habits, including

increased consumption of dairy products and/or calcium rich vegetables" [18].

Varieties of Fruit Juice Consumed

According to juice manufacturers' data, reported by Smith and colleagues, 50% of the fruit juice consumed by children under 5 years of age was apple juice [8]. In the study of 4- and 5-year old Latino children, 80% of the fruit juice consumed was reported as orange juice and 20% was reported as apple juice [10]. In the study of 2- and 5-year old rural children, 39% of the fruit juice consumed was reported as "mixed fruit juice" [11]. Apple juice is the first juice listed on most mixed fruit juices. Thirty percent of the juice consumed was plain apple juice, 23% was orange juice, 7% was grape juice, and 1% was pear or pear-apple juice (Fig. 1).

Excess Fruit Juice Consumption

In a population-based study of healthy young children, the distribution of fruit juice consumption was skewed to the right (Fig. 2). The mean fruit juice intake was 5.5 fl oz/day. Eleven percent of children consumed \geq 12 fl oz/day of fruit juice and 2% consumed \geq 16 fl oz/day of fruit juice [11]. These percentages are similar to those reported by juice manufacturers for infants, with a mean fruit juice intake of 5.0 fl oz/day and 1% of the infants consuming more than 21 fl oz/day [19]. Consumption of \geq 12 fl oz/day of fruit juice by young children has been defined as excessive juice intake for several reasons. This amount of juice is also approximately twice the average intake of young children in several population-based studies [10,11,20]. Twelve fl oz/day of fruit juice equals the number of fruit servings recommended for young children (2 servings/day) and is 50% of the fruit servings recommended for those with higher caloric intakes (4 servings/day) [13]. This amount of juice is also the maximum amount recommended for young children by several experts [9,20].

Children drinking excessive amounts of fruit juice (\geq 12 fl oz/day) had much higher intakes of simple carbohydrates than children drinking less fruit juice; 36.5% vs 28.7%, ($p < 0.0001$)

Fruit Juice Consumed

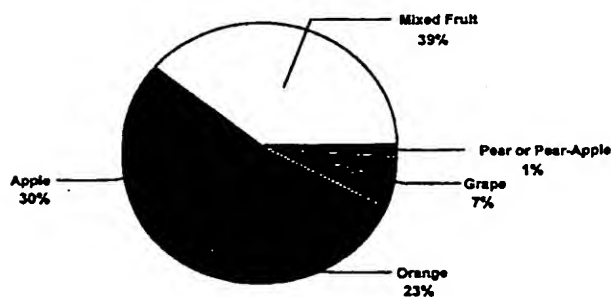


Fig. 1. Varieties of fruit juice consumed by young children.

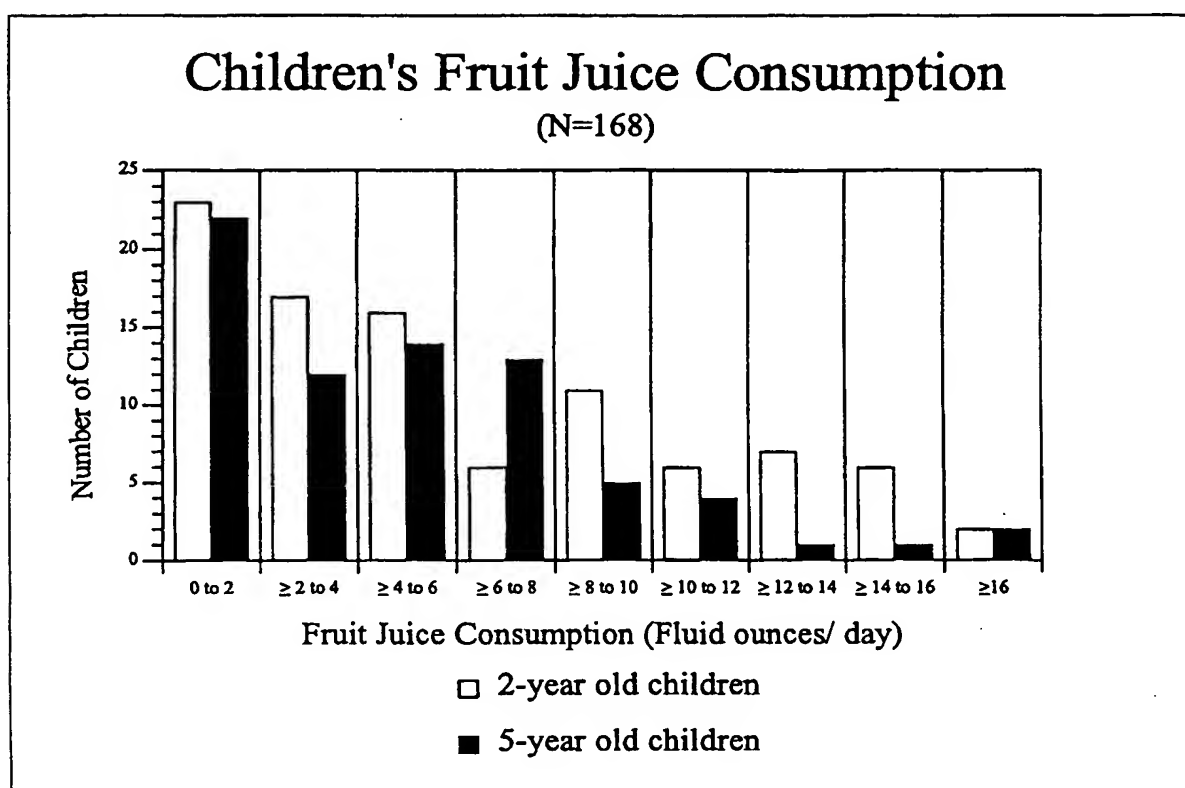


Fig. 2. Distribution of children's fruit juice consumption (7-day mean), shown separately for 2- and 5-year old children. Reprinted, with permission, from Dennison BA, Rockwell HL, Baker SL. Pediatrics, in press, 1996.

Table 1. Fruit Juice Composition (Per 8 Fluid Ounce Serving)

	Apple	Orange	Grape	Pear
Energy (kcal)	117	112	154	196
Fructose (g)	13.9	11.5	21.0	21.3
Glucose (g)	6.2	13.2	17.2	6.3
Sucrose (g)	4.2	1.7	0.0	6.0
Sorbitol (g)	0.6 to 1.2	0.0	0.0	4.5 to 5.5
Total fiber (g)	0.3	0.8	1.3	11.6
Soluble fiber (g)	0.1	0.3	0.5	4.3
Pectin (g)	0.1	0.5	0.5	1.8

All data, except sorbitol, are from the Minnesota Nutrition Data System; Program Version 2.6; Food Data Base Version 8A; Nutrient Data Base Version 23 (Minneapolis, MN), which is derived from the USDA, food manufacturers, foreign food tables, and scientific literature.

Sorbitol data are from USDA, Provisional Table on the Sugar Content of Selected Foods, Nutrient Data Research Branch, Nutrition Monitoring Division, October 1986 (HNIS/PT-105); and Lee HS and Wrolstad RE: Apple juice composition: sugar, nonvolatile acid, and phenolic profiles. J Assoc Off Anal Chem 1988; 71:789-794.

and 34.2% vs 28.4% ($p < 0.05$) for the 2- and 5-year old children, respectively [11]. The major naturally occurring sugars present in fruit juices are fructose and glucose (Table 1). Thus, it is not

surprising that fructose intake was twice as high and glucose intake was 80% higher among the children drinking ≥ 12 fl oz/day of fruit juice compared to children drinking less juice [11]. Children drinking excessive fruit juice consumed a significantly lower percentage of total calories from total fat (29.1% vs. 34.0% [$p < 0.0005$] and 28.0% vs. 33.2% [$p < 0.05$] for the 2- and 5-year old children, respectively) and saturated fat (12.3% vs. 13.9% and 11.1% vs. 12.3% [both $p < 0.05$] for the 2- and 5-year old children, respectively) than children drinking less juice [11]. Two-year old children who consumed excessive amounts of fruit juice (≥ 12 fl oz/day of fruit juice) also had higher energy intakes, when standardized by either weight or height, than children who consumed less fruit juice; 47.1 vs. 41.9 kcal/kg and 1528 vs 1371 kcal/m, respectively (both $p < 0.05$) [11].

PREFERENCE FOR SWEETENED BEVERAGES

Preference for sweetened beverages is present even in neonates [21]. Studies in newborn infants have demonstrated a preference for, and increased consumption of, sweetened water compared to plain water [21]. Moreover, the consumption of

Fruit Juice Consumption

sweetened beverages by infants increases proportionate to the sugar concentration and to the sweetness of the sugar [22]. Preschool-aged children's food preferences are best explained by sweet taste and exposure [23,24], so it is not surprising that most young children drink naturally sweet fruit juice (90% by the age of 1 year) [8], and that some children, if given the opportunity, might consume excessive amounts of naturally-sweet fruit juice. Rats offered sugar-sweetened water in addition to rat chow consumed more calories and gained more body weight than rats fed just rat chow and plain water [25]. The rats decreased their chow intake, but not enough to offset the calories provided by the sugar solution.

FRUIT JUICE AND DENTAL CARIES

Concerns about fruit juice consumption and dental caries have been raised by a number of professional groups since at least 1978. The AAP and the American Academy of Pedodontics issued a joint statement concerning fruit juice in baby bottles and the development of nursing-bottle caries [26]. They stated that fruit juices should be introduced when the infant is ready to drink from the cup and that "the use of juices from a bottle should be discouraged." The AAP Committee on Nutrition has cautioned that juice in bottles, when used as a pacifier, and prolonged bottle feeding predisposes to nursing bottle caries [7]. Despite this, \$164 million was reportedly spent in 1992 on bottled baby juice [9]. Dental caries is the most prevalent disease known in the US. According to Healthy People 2000, 53% of children, aged 6 through 8 years in 1986-87, had one or more dental caries [1].

FRUIT JUICE AND NON-SPECIFIC DIARRHEA OR "TODDLER'S" DIARRHEA

The role of juice carbohydrate malabsorption in chronic nonspecific diarrhea in children has been recognized for some time [27,28]. In 1991, the AAP Committee on Nutrition issued a statement concerning sorbitol, a naturally occurring but non-absorbable sugar alcohol present primarily in pear juice and apple juice [19]. The Committee cautioned that the "excessive use of fruit juice" may result in gastrointestinal symptoms, such as chronic diarrhea, abdominal pain, or bloating. Fructose malabsorption is relatively common [29] and increases at higher concentrations and at higher doses of fructose [30]. In the presence of sorbitol, fructose malabsorption is further increased [31]. When combined with glucose, however, fructose malabsorption decreases, and at equal concentrations of fructose and glucose, fructose is rarely malabsorbed [30]. Fruits and their corresponding juices differ by nutrient density, carbohydrate composition and concentration, and the amounts of sorbitol, pectin, and fiber present (Table 1). These differences affect

gastric emptying time and result in varying degrees of carbohydrate malabsorption and gastrointestinal symptoms. Hydrogen breath testing is a noninvasive method to measure bacterial fermentation of malabsorbed carbohydrate and other fermentable substances reaching the colon [32]. A recent paper, using this technique, reported that significantly more infants and toddlers had evidence of malabsorption following ingestion of apple juice compared to white grape juice; 54% vs 19% had a hydrogen breath test > 20 ppm (Fig. 3) [8].

EXCESS FRUIT JUICE CONSUMPTION AND DECREASED WEIGHT AND/OR HEIGHT

Among children referred for evaluation of failure to thrive, excessive fruit juice consumption (12 to 30 fl oz/day) was reported as a contributing factor in nonorganic failure to thrive in eight children, aged 14 to 27 months [33]. In each case, deterioration of weight and linear growth progression coincided with the excessive juice consumption. All eight children had weights < 5 th percentile for age and sex and 5 also had lengths ≤ 5 th percentile for age and sex [34]. The children's diets were hypocaloric, providing 78% to 92% of the recommended energy intake for age and weight. With a marked reduction in the amount of juice consumed, an increase in the total calories consumed, and adoption of a more balanced diet, the children gained weight and the growth failure resolved. Breath hydrogen testing revealed malabsorption of fructose and/or sorbitol.

In a population-based study, the prevalence of children with decreased stature (height < 20 th percentile for age and sex) [34] was higher among children who consumed ≥ 12 fl oz/day of fruit juice than children consuming less juice [11]. Forty-two percent of children consuming ≥ 12 fl oz/day of fruit juice had decreased stature compared to 14% of children drinking < 12 fl oz/day ($p < 0.01$). After statistical adjustment for maternal height, child age, child sex, and child age-sex interaction, children consuming ≥ 12 fl oz/day of juice were found to be

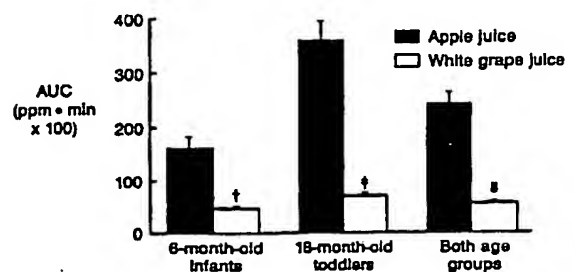
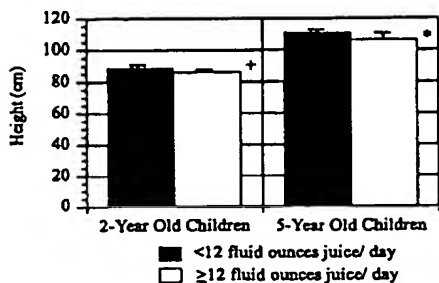


Fig. 3. Integrated breath H_2 excretion following juice consumption. * Mean \pm SEM. Comparisons are between apple juice and white grape juice. $\dagger p < 0.05$; $\ddagger p < 0.005$; $\S p < 0.001$. (Reprinted, with permission, from Smith MM, Davis M, Chasalow FI, Lifshitz F. Pediatrics 95:343, 1995.)

Child Height vs Juice Consumption



General linear models were used to statistically adjust child height for maternal height, child age, child sex, and child age-sex interaction. Data shown are least squares mean \pm 95% confidence interval. * $p < 0.05$ + $p < 0.005$

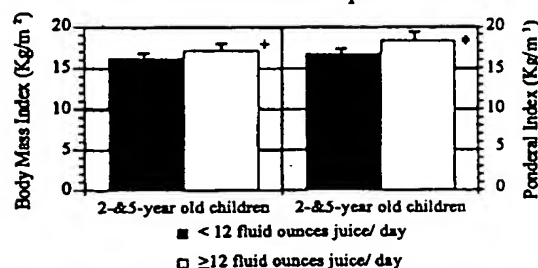
Fig. 4. Adjusted height of children consuming < 12 fl oz/day and ≥ 12 fl oz/day of fruit juice, shown separately for 2- and 5-year old children. General Linear Models was used to adjust child for maternal height, child age, child sex, and child age-sex interaction. Figure developed with permission, from data presented in Table 5, from Dennison BA, Rockwell HL, Baker SL. Pediatrics, in press, 1996.

significantly shorter than children consuming < 12 fl oz/day (Fig. 4). The adjusted child heights were 86.5 vs 89.3 cm ($p < 0.005$) and 106.5 vs 111.2 cm ($p < 0.05$) for the 2- and 5-year old children, respectively. In this study, no information was collected regarding gastrointestinal symptoms that might accompany malabsorption. Many parents might not recognize gastrointestinal symptoms accompanying carbohydrate malabsorption in young children [27], as only a small fraction (10% in a recent study) of children with evidence of malabsorption (hydrogen breath test > 20 ppm) had gastrointestinal symptoms (e.g., diarrhea) [8].

EXCESS FRUIT JUICE CONSUMPTION AND OBESITY

In a population-based study, the prevalence of overweight children was higher among the children consuming ≥ 12 fl oz/day of fruit juice [11]. Fifty-three percent of children drinking ≥ 12 fl oz/day of juice had a body mass index (BMI) ≥ 75 th age- and sex-specific percentile [36] compared to 32% of those drinking < 12 fl oz/day ($p = 0.06$). Thirty-two percent of children drinking ≥ 12 fl oz/day of fruit juice had a BMI ≥ 90 th age- and sex-specific percentile compared to 9% of those drinking < 12 fl oz/day ($p < 0.01$). Thirty-two percent of children drinking ≥ 12 fl oz/day of fruit juice had a ponderal index ≥ 90 th age-specific percentile compared to 5% of those drinking less juice ($p < 0.005$). After statistical adjustment, for maternal height, child age, child sex, and child age-sex interaction, the children who consumed ≥ 12 fl oz/day of fruit juice were significantly more overweight than children consuming less juice, with a mean BMI of 17.2 vs 16.3 kg/m² ($p < 0.005$) and a mean ponderal index of 18.4 vs 16.8 kg/m³ ($p < 0.0001$) (Fig. 5).

Child Body Mass Index and Ponderal Index vs Juice Consumption



General Linear Models were used to statistically adjust child body mass index and ponderal index for maternal height, child age, child sex, and child age-sex interaction. Data shown are least squares mean \pm 95% confidence interval. * $p < 0.005$ + $p < 0.0001$

Fig. 5. Adjusted body mass index and ponderal index of children consuming < 12 fl oz/day and ≥ 12 fl oz/day of fruit juice. General Linear Models was used to adjust child body mass index and ponderal index for maternal height, child age, child sex, and child age-sex interaction. Figure developed with permission, from data presented in Table 5, from Dennison BA, Rockwell HL, Baker SL. Pediatrics, in press, 1996.

DISCUSSION

Fruit juice consumption has been increasing over the past 20 years and is likely to continue. Approximately 50% of all servings of fruit eaten by children, aged 2–18 years, are consumed as fruit juice and one-third of all fruits and vegetables consumed by preschool-aged children are consumed as fruit juice. Thus fruit juice consumption contributes significantly to total fruit and vegetable intake of children, especially young children. With the increased emphasis on eating more fruits and vegetables, combined with increased promotion of fruit juices as healthy, low-fat, nutritious beverages and snacks, fruit juice consumption is expected to continue to increase. Although recent data suggests an increase in the mean and median amounts of fruit juice consumed by young children, there is little data available to determine if the number of young children drinking excess amounts of juice is also increasing. Anecdotal reports by Head Start and other daycare providers, pediatric care providers, and pediatric gastroenterologists suggest that the number of young children consuming excessive amounts of fruit juice has been increasing over the past 5–10 years (personal communication). The consumption of fruit juice by young children in bottles continues to be of concern. Despite numerous recommendations to the contrary, the juice bottle toting toddler is commonly seen in pediatric waiting rooms and grocery stores. The fact that 50% of US children, aged 1 through 5 years, do not meet the RDA for calcium is alarming. Whether the per capita decline in calcium and milk consumption is related to the increased availability and consumption of other beverages such as fruit juice and/or soda pop is purely speculative but plausible.

Excessive fruit juice consumption has been associated in some children with failure to thrive and/or decreased height.

Children drinking excessive fruit juice consumed a greater proportion of total calories from simple sugars (twice as much fructose and 80% more glucose) than children drinking less juice. For the most part, simple sugars are "empty calories." Some of the excess juice drinkers might be substituting the extra juice for more nutritious foods, while other children might be malabsorbing some of the carbohydrates or sugars (especially fructose and/or sorbitol) found in the fruit juice. Both of these factors could adversely affect growth, contributing to the development of decreased weight and/or height.

In other children, excessive fruit juice consumption has been linked to obesity. Analogous to animal studies [25], it is possible that some children, when offered sweetened beverages in addition to food, will consume extra calories and gain excess weight. The higher energy intake, standardized by either weight or height, of 2-year old children consuming excessive fruit juice supports this theory. In other words, the excessive juice consumption contributes to excessive caloric consumption which results in extra weight gain.

Since infants and young children prefer sweetened beverages, it is not surprising that some infants and children, if given the opportunity, might drink excessive amounts of naturally sweet fruit juice. Some parents might offer children juice instead of water when they are thirsty, or could give the children too much juice to drink for any number of reasons, including convenience, ready availability, favorable cost, perceived health benefits, and the child's preference for fruit juice. Children's eating behaviors are also influenced by parents and caretakers, social and environmental influences (such as the use of foods as rewards and the withholding of food as punishment), and media influences.

CONCLUSIONS

The recent studies linking excessive fruit juice consumption to decreased weight and/or height in some young children, and to obesity in other children are cause for concern. They, of course, need to be replicated in additional populations of children. Longitudinal studies are needed to evaluate causality. As with most things in life, however, moderation is probably best. Until further studies prove otherwise, it seems prudent for parents and caretakers to limit consumption of fruit juice by young children to < 12 fl oz/day.

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WHY IS EVERYONE GOING ON ABOUT CHILDHOOD OVERWEIGHT AND WHAT CAN WE DO ABOUT IT?

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One of this decade's dominant health issues relates to the fact that Australian children and adolescents are growing fatter at an alarming rate. Understanding why this is so, why things have changed, why being overweight matters, and most importantly, what we can do to prevent it form the foci of this article.

WHAT DO WE MEAN BY OVERWEIGHT AND OBESE?

How do we know if someone is overweight? This is a good question, particularly as most fatness in children is actually not noticed by adults at all. The degree of fatness is determined by calculating a child's Body Mass Index, which is their weight in kilograms divided by their height in metres squared. $BMI = \text{weight(kg)} / \text{height (m}^2\text{)}$.

The Body Mass Index (BMI) is then compared to cut-off points for ages (Cole, T.J. 2000) as shown in Table 1.

Table 1: BMI cut-off points for overweight and obesity

Age	BMI defining overweight		BMI defining obesity	
	Boys	Girls	Boys	Girls
2-year-old	≥ 18.41	≥ 18.02	≥ 20.09	≥ 19.81
5-year-old	≥ 17.42	≥ 17.15	≥ 19.3	≥ 19.17
10-year-old	≥ 19.84	≥ 19.86	≥ 24	≥ 24.11
15-year-old	≥ 23.29	≥ 23.94	≥ 28.30	≥ 29.11
≥ 18 years	≥ 25	≥ 25	≥ 30	≥ 30

Source: Cole, T.J. 2000.

So, for example, if a 10-year-old boy weighs 45 kilograms and is 140cm tall, his BMI would be: $BMI = 45 / (1.4)^2 = 22.9$. As you can see above, the cut-off point defining overweight for a 10-year-old boy is ≥ 19.84 and therefore, this boy would be considered to be overweight.

It is interesting for us to understand that most overweight in children is not recognised by adults. This suggests that adults are not very sensitive to fatness in children and/or that we tend to expect that children will have phases when they are a little 'chubby' or are carrying 'puppy fat'. The

expectation has been that this 'puppy fat' will disappear as the child grows older—however, the fact is that once we gain body fat it is very difficult to lose it again. This is one of the reasons why it is important to prevent unneeded weight gain in the first place.

STUDENT ACTIVITIES

- a. Name and describe the measurement that is used to determine the degree of 'fatness' of a person?
b. Use the data in Table 1 to find the cut-off points for overweight and obesity for 15 year old males and females.
c. Use your text book or other resources to find definitions of obesity and overweight that do not rely on BMI calculations.
2. Dr Campbell makes the observation that 'most overweight in children is not recognised by adults'. Provide possible reasons for this lack of recognition by adults.

SO, HOW BIG IS THIS PROBLEM? HOW MANY AUSTRALIAN CHILDREN ARE OVERWEIGHT?

Table 2: Increase in rates of overweight and obesity in 10 years

Age	Boys			
	7-11 years		12-15 years	
BMI grade	1985	1995	1985	1995
Overweight	9.7	11.6	8.8	20.0
Obese	1.5	3.7	1.9	6.1
Overweight/obese	11.2	15.3	10.7	26.1

	Girls			
	7-11 years		12-15 years	
BMI grade	1985	1995	1985	1995
Overweight	11.0	17.2	10.1	14.5
Obese	1.9	6.3	1.3	4.4
Overweight/obese	12.9	23.5	11.4	18.9

Source: Magarey et al, 2001.

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The rates of childhood fatness have increased greatly over the past 20 years. Indeed, Australia has one of the highest rates of childhood fatness in the world. We know that between 1985 and 1995 the level of combined overweight and obesity has more than doubled. So, in 1995, 21 per cent of boys aged 2–17 years were overweight or obese and 23 per cent of girls. At the moment childhood obesity in Australia is rising at a rate of 1 per cent per year, a trend which suggests that, if nothing changes, half of all Australian children will be overweight by the year 2025.

STUDENT ACTIVITY

3. Using the data in Table 2 compare and contrast trends in obesity and overweight in:
 - a. age groups 7–11 and 12–15
 - b. boys and girls

DOES BEING OVERWEIGHT REALLY MATTER ANYWAY?

It is easy to think that we're making a big deal out of just a little 'puppy fat'. It's also easy to think that we are worrying about body fatness because the 'ideal' image of a healthy and attractive person is slim, not fat; just take a look in any magazine and you'll see that most models, actors and celebrities are slim. Yet, the fact is that our concern has nothing to do with how we look, and everything to do with how children will feel, today and into the future. The reality is that fat, no matter what we call it, is not good for our health.

PHYSICAL HEALTH PROBLEMS

On top of the social issues affecting overweight children and adolescents there are a whole lot of effects on a child's physical health. These are outlined in Table 3 below.

One very important problem with becoming fat when we are young is that the fatness is very likely to continue on into adolescence and even into adulthood. For example, about 80 per cent of obese adolescents will become obese adults. It is very difficult to lose weight.

In addition to the risk of illness in childhood, it also seems that being obese as a child or adolescent increases the risk of a range of diseases and disorders in adulthood, even if you become a normal weight adult. Together these things highlight just how important it is that overweight and obesity are stopped before they start—prevention is the key.

Table 3: Health consequences associated with childhood obesity

High prevalence	Intermediate prevalence	Low prevalence
<ul style="list-style-type: none"> – social problems – fast growth and early development (girls) – continuation of fatness into adulthood – abnormal blood fats 	<ul style="list-style-type: none"> – fatty liver – abnormal glucose metabolism (relates to adult onset diabetes) 	<ul style="list-style-type: none"> – problems with bones e.g knees, ankles, feet, hips – sleep apnoea (disturbed sleep patterns) – polycystic ovary syndrome – gall bladder disease – high blood pressure – type 2 diabetes

Source: World Health Organization, 1999.

PSYCHO-SOCIAL PROBLEMS ASSOCIATED WITH FATNESS IN CHILDREN

One of the very important problems for children and adolescents who are overweight or obese is that their weight is very likely to impact on how they feel about themselves and on how they interact with other children. Being overweight as an adolescent may affect levels of confidence and self-esteem.

STUDENT ACTIVITY

4. a. Make a list of the health consequences associated with obesity in childhood and adolescence. Include consequences for physical, social and emotional health.
- b. The author states that in relation to overweight and obesity 'prevention is the key'. Apart from the risk of illness in childhood, list the other reasons outlined in the article as to why overweight and obesity should be prevented.

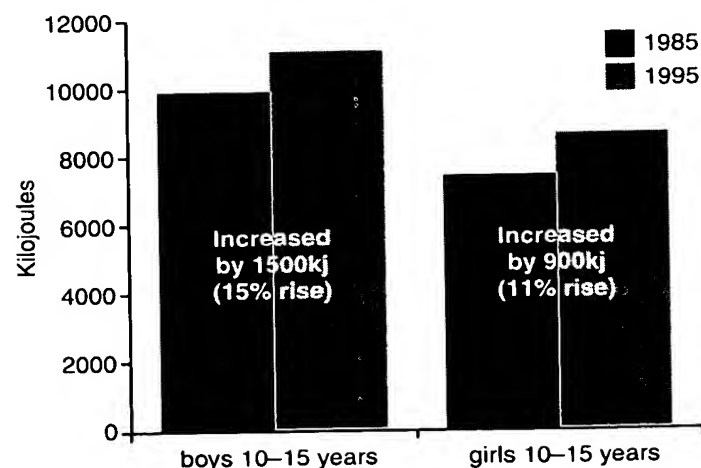
SO WHY ARE CHILDREN BECOMING OVERWEIGHT?

CHANGES TO OUR DIET

The simple fact is that children, like adults will store the kilojoules from food, which are not used in growth and activity, as fat on their bodies. So, if we eat more than we use up we deposit this as fat. This sounds very simple; however, knowing how much food to eat and how much activity to do can be very challenging. Understanding how Australian children's and adolescents' diets have changed over the past 15 years provides us with some interesting clues.

Over the 10-year period from 1985 to 1995 (see Figure 1) the diets of Australian children and adolescents changed a lot—and this happened at the same time as they started to grow fatter (Cook, T., Rutishauser, L. & Seelig, M. 2001). Overall, the kilojoule intake of 10–15-year-old girls has increased by 11 per cent, which means by 900kJ per day, and in boys by 15 per cent—a kilojoule increase of around 1500kJ per day, over this time. As mentioned before, increased intakes of energy, if they are not compensated for by increased activity (energy expenditure) will be stored as body fat.

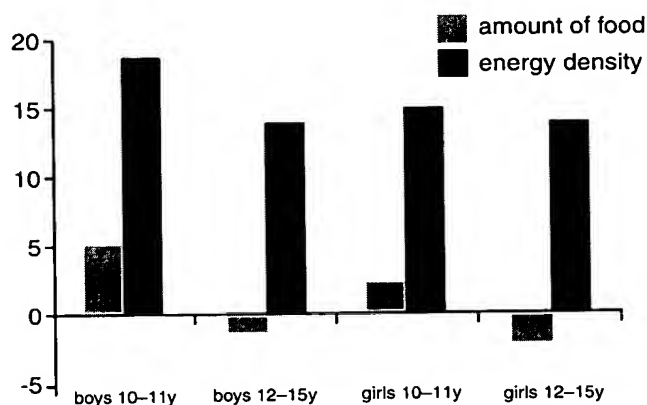
Figure 1: Changes in energy intakes between 1985 and 1995



Cook P, Rutishauser LHE, Seelig M (2001). *Comparable data on food and nutrient intake and physical measurements from 1983, 1985 and 1995 national surveys*. Commonwealth Department of Health and Aged Care, Canberra

You might be tempted to assume that they are all eating more food than they did before, yet, this seems not to be the case. In fact, as shown in Figure 2 below, it seems the amount of food children and adolescents eat has remained about the same, what has changed is the type of food they are eating and the number of kilojoules this food contains. This graphic shows that the energy density of the diet has increased by around 14–18 per cent.

Figure 2: Per cent change in amount of food and energy density of food 1985 to 1995



Cook P, Rutishauser IHE, Seelig M (2001), *Comparable data on food and nutrient intake and physical measurements from 1983, 1985 and 1995 national surveys*, Commonwealth Department of Health and Aged Care, Canberra

Table 4: High and low energy density choices of some commonly-consumed items

Food type	High energy density choice	Lower energy density choice
Drinks ¹	Cola or lemonade ~ 500kJ/250ml	Water 1 cup = 0kJ per 250ml
Sweet snacks	Iced doughnut = 1360kJ per serve (70gm)	Apricot Fruit Bread ² = 670kJ per serve (2 slices)
Sweet biscuits	Chocolate coated and cream filled biscuit ~400kJ per biscuit	Snack Right Fruit Slice ² ~155kJ per biscuit
Sweet 'treat'	Chocolate covered bar (e.g. Mars Bar or Kit Kat) ~2000kJ/100gm	Jelly snakes, jelly lollies ~1520 kJ per 100gm
Ice-cream	Magnum Classic ~1200kJ/serve	Frozen yoghurt/Calipo lcpole ~365kJ per serve
Dry Biscuits	Savoury Shapes ~2050 kJ /100gm	Vita Wheats ² ~1640kJ per 100gm
Salty snacks	Potato crisps/ corn chips ~2100 kJ/100gm	Pretzels or rice crackers ~1619-1670kJ per 100gm
Potato	Potato chips 835kJ/150gm	Potato, steamed/ boiled or baked with no fat ~290kJ/150gm

NOTES

¹ Soft-drinks provide a good example of the problems of 'super sizing'. For example, a can is 375ml, containing 675kJ while a bottle is now 600ml containing 1080kJ. We know that children and adults consume more when the serving size is increased.

² Also high in fibre and have a lower glycaemic index (means you don't get hungry so quickly after eating)—both important in a health promoting diet.

To understand this is important to understand the concept of the energy density of a food. This refers to the kilojoules contained in a certain weight of food. Table 4 shows some examples of how the energy density of similar types of foods can be very different, and helps us to understand how it is that our kilojoule intakes have increased over the past few years.

STUDENT ACTIVITIES

- In the article there are two main reasons given for the increase in overweight and obesity in children and adolescents. Explain these reasons.
- Describe the change in kilojoule intake in Australian children and adolescents between 1985 and 1995.
 - The author suggests that the increase in kilojoule intake is not due to the amount of food but to the energy density of food. Define energy density.
- Identify two trends in Figure 2: 'Per cent change in amount of food and energy density of food 1985-1995'
- From Table 4, choose three examples of high energy density choice foods and their lower energy density equivalent.
 - What observations can you make about the differences in composition between the higher energy density choices and the lower energy density choices? Use the following terms in your observations—fat, sugar, dietary fibre, processed, unprocessed.

When we look at the kinds of foods that have changed in children's and adolescent's diets over the time period between 1985 and 1995 we can see that the changes are all in foods which would have increased the energy density of the diet. What we saw was:

- 40-56 per cent increase in confectionary eaten
- 29-48 per cent increase in soft drink, cordial and juice consumption
- 59-136 per cent increase in sugar products and dishes, e.g. sugar, honey, jam and chocolate spreads, dishes other than confectionary where sugar is the major component
- 46 per cent increase in cereal-based products, e.g. cakes, buns, biscuits, pastries and sweetened breakfast cereals.

CHANGES TO OUR ACTIVITY

At the same time as we have seen changes in Australian children's and adolescent's eating, we have seen changes to the amount of physical activity they do. Research shows for instance that far fewer children now walk or cycle to school. We have seen changes in the number of activities that encourage them to sit still and also use very little energy. In recent years we have seen:

- access to pay television increase from 5 per cent in 1996 to 19 per cent in 2000 (AC Nieslen Media International 2001)
- an increase in home computer use with 82 per cent of adolescents in Australia having a home computer in 2001 (an absolute increase of 13 per cent since 1999)
- increases in the numbers of households who own two or more television sets (61 per cent in 2002)
- increases in the number of households to 87 per cent in 2001 who own one or more video cassette recorders or VCRs (AC Nieslen Media International 2001).

Together, this rapid increase in interesting and enticing opportunities to sit still seems likely to have promoted a more sedentary lifestyle but we can't be sure of this. It may be that these sedentary pursuits now just replace other sedentary hobbies like reading.

HOW MUCH IS ENOUGH?

Whatever the case, it remains important that we all understand just when we have spent enough time sitting still at our screens. The Australian Physical Activity Recommendations suggest that children and adolescents spend no more than two hours per day watching television and using other electronic entertainment media.

When we compare this to what is actually happening we can see that there is a lot of room for improvement:

- on average, Australian children and adolescents watch around two-and-a-half hours of television per day
- about two-thirds of children currently watch more than two hours of television per day
- more than one-quarter actually watch more than four hours per day.

IN SUMMARY

This article has discussed that:

- child and adolescent weight has increased dramatically over the past 10–20 years
- this has important health consequences for the child now but also in the future
- there are good explanations of why it is that body weight has increased so rapidly
- Australian children's diets have changed dramatically, with kJ intake increasing by 11–15 per cent primarily because the energy density of the food they choose has increased
- Australian children and adolescents appear to be less physically active than they were
- Australian children and adolescents now have more opportunities to be sedentary than ever before
- if we understand the causes of increased body weight, we have increased opportunity to stop this occurring.

CAUSES OF OBESITY

Obesity in children may be caused by:

- genetics or an abnormal endocrine gland—it is thought that genes may play a role in between 25 to 40 per cent of all cases of obesity
- eating more kilojoules than are used—children, like adults, will store fat in if they eat more energy (kilojoules) than they use
- lack of physical activity—Australian children are less active than they were in the past
- sedentary pursuits—Australian children watch, on average around two-and-a-half hours of television a day, as well as spending time using computers and other electronic games. It is likely that less sedentary activities are being replaced by more sedentary ones.

FOOD AND ACTIVITY CHOICES WE MAKE EVERYDAY ARE IMPORTANT

Try to make healthy choices:

- don't buy soft drink or cordial and limit fruit juice to one glass a day

- drink unflavoured water and switch to low-fat milk
- choose nutritious snacks, such as fruit, yoghurt and sandwiches
- start the day with a filling, nutritious breakfast and switch to a low fat, low sugar, whole wheat or oat breakfast cereal
- reduce the number of takeaway meals you eat and try to choose takeaway foods that are lower in kilojoules
- be wary of foods marketed as low fat as these are usually high in sugar and still high in kilojoules
- find ways to incorporate physical activity into your routine, e.g. allowing time to walk to school rather than driving, and making time for being active by switching off the television.

STUDENT ACTIVITIES

7. a. Identify the types of foods that have shown considerable increase in consumption by children and adolescents between 1985 and 1995.
b. Suggest reasons for the increases in foods identified in part a.
c. Identify the changes in recent years that may have promoted a more sedentary lifestyle, as outlined in the article.
d. Provide reasons why the author has been careful not to conclude a causal relationship between the changes identified in part c) and the increase in overweight and obesity.
8. 'If we understand the causes of increased body weight, we have increased opportunity to stop this occurring'. Explain this statement using information in the article. Include a description of some of the causes of increased body weight and methods for prevention.

Going Further

1. Foods listed in Table 4 are generally described as 'indulgence' or 'treat' foods. The lower energy density choices are not necessarily 'nutrient dense'. Use your textbook or other resources to define the term 'nutrient density' and to provide examples of snack choices that are high in nutrient density and medium to low in energy density.
2. a. Use the internet and other resources to research current strategies being undertaken in your state that are designed to help prevent and reduce overweight and obesity in childhood & adolescence. Name and describe the strategies.
b. Work in small groups to complete this activity. You have been asked by the local primary school to develop a strategy to help prevent or reduce overweight and obesity for its students. Use the information in the article and from reputable websites to plan the strategy. The Ottawa Charter may assist in your planning and evaluation. Describe components of the strategy including the target group and resources required. Compile a list of criteria that you would use to evaluate the success of your strategy?
3. The guidelines listed are for good health in general, not just to reduce the risk of overweight and obesity.
a. Research and make a list of other health problems or diseases for which the risk would be reduced if the guidelines in were followed.
b. For each of the guidelines give yourself a score out of 3 where 3 = always, 2 = mostly, 1 = sometimes, 0 = never.
c. Use your scores to choose 3 areas for improvement in your lifestyle. Make practical suggestions as to how you could make the changes. Put them into action.

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- World Health Organisation, Obesity: preventing and managing the global epidemic: Report of the WHO consultation. 1999, World Health Organisation: Geneva.

USEFUL WEBSITES

- About overweight and obesity [online], Population Health, Australian Department of Health and Ageing
<www.health.gov.au/pubhlth/strateg/hlthwt/obesity.htm>
- Australasian Society for the Study of Obesity <www.asso.org.au/home>
- Australian Guide to Healthy Eating
<www.health.gov.au/internet/wcms/Publishing.nsf/Content/health-pubhlth-publicat-document-fdbrox-cnt.htm>
- Childhood and adolescent obesity [online], Healthy Eating Club
<www.healthyeatingclub.com/info/articles/infant-child/childhood-obesity.htm>. Heart Foundation <www.heartfoundation.com.au/>
- Kinect Australia—Vicfit <www.vicfit.com.au/>
- The Parent's Jury <www.parentsjury.org.au/tpj_index.asp>

DO YOU KNOW WHAT YOU ARE REALLY DRINKING?

by Jane Duthie

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INTRODUCTION

Social, economic and cultural factors strongly influence individual choices about diet and physical activity. With so much talk about the obesity epidemic from health experts, the government and the media, it has hopefully made us more aware of the extra kilojoules we are consuming in the high fat and high sugar foods being produced. Although many Australians would like to adopt a healthier lifestyle, many encounter substantial barriers that make it difficult to follow diet and activity guidelines. The increase in obesity, behavioural problems and physical inactivity is of particular concern for a number of groups, in particular young children and adults who are establishing lifelong behavioural patterns that affect their physical, social and emotional health status.

In 2006, a display of 18 drinks posters for the staff and students at John Forrest Senior High School was produced. Each A3-size laminated poster included a photo of the drink, a table of nutritional information found on the label for 100g serves, nutritional information for the container size instead of the serving size, other information found on the drink label, and a list of ingredients and additives that were colour-coded as white, green, orange and red (see Table 2) with their possible adverse effects to health. The display was made even more visual with the actual drinks and a see-through container showing the amount of sugar in each container of drink beside each poster. The focus of the display was originally to be the amount of sugar in each of the drinks and how this might have an affect on the physical health status, and also the behaviour of the students in our school. Many choose to consume large amounts of sweetened drinks during the school day because they have been readily available through the school canteen. Some also bring large bottles of soft drink in their bags from home or purchased on the way to school.

WHAT IS THE PROBLEM WITH SOFT AND FRUIT DRINKS?

- most contain high amounts of sugar, some caffeine, acids or preservatives
- high concentrations of sugar can be harmful to teeth causing decay
- sugar is considered to be an 'energy dense' food
- soft and fruit drinks have become a staple drink for many children and young adults
- people do not increase their physical activity to burn up the extra kilojoules
- soft drinks are now the most purchased items in Australian supermarkets
- soft drinks have practically no nutritional value
- some soft drinks may contain inappropriate additives that are possibly hazardous to health for some individuals
- soft drinks are not considered part of a healthy diet
- many drink containers provide up to three serves, but are consumed as one serve
- drinks are marketed in an appealing way to children and adults alike.



THE PROBLEM WITH SUGAR

Sugar has been listed in the orange coded additives because it has an affect on our health if consumed in larger quantities than recommended in nutrition guidelines. Energy-dense sugar foods are not a part of a healthy eating plan. The Australian Guide to Healthy Eating recommends these foods should be chosen sometimes and in small amounts. A total of 1–3 serves per day is the allowed amount of extra foods for adolescents. Two tablespoons of sugar equals one serve of these extra foods, about 600 kilojoules. This includes not only the sugar content of drinks but sugars found in other food products consumed, such as that added to tea and coffee, donuts, jams, biscuits, cakes, ice-cream, canned fruits, chocolates and confectionary. Don't forget the hidden sugar in sauces, even tomato sauce and in breads, yoghurts, custards, spreads and hundreds of other foods. The 1–3 serves of 'extra' foods also include the energy value in foods from added fats.

The World Health Organization recommends reducing your intake of added sugars to less than 10 percent of your total energy intake. That means, if you eat 10,500 kilojoules a day (about the average requirement for a normal 16-year-old female) you should eat less than 16 teaspoons of sugar from all food and drink sources. One can of soft drink contains about 10 teaspoons of sugar. A report from the International Obesity Task Force (2006), based on a study at Boston's Children's Hospital, concluded that a single can of sweetened drink a day could result in a weight increase of 450 grams every three to four weeks or about 6 kilograms a year.

In recent years food and drink portions have increased in size. They began to increase in the 1970s, rose sharply in the 1980s and have continued to increase along with increasing body size. Although recognised as a problem

in the fast food industry other food manufacturers are also increasing food and drink portions. Large portion sizes and energy-dense foods are used extensively in marketing in restaurants, supermarkets and food companies. Many drinks now come in 600ml containers, where once they were less than 400ml and originally only about 250ml.

Serving size should be determined by body size and the amount of daily physical activity. Bigger people need more food; smaller people need less food. More active people can consume more kilojoules; less active people need to restrict their intake of energy-dense foods such as sugar. When looking for sugar in the list of ingredients, don't forget to look for all the other common names such as sucrose, glucose, dextrose, golden syrup and maple syrup.

Table 1 below compares the amount of sugar in 100ml of each drink as stated on the labels. Each drink container provides the amount of sugar in a serving size. However, not many individuals will drink a half or one-third of the container of drink because the manufacturer states that as the recommended size of the serve. People will more often consume the whole of a container of drink and therefore in most cases an even greater amount of sugar and therefore, kilojoules.

From the table it would appear that those drinks with less sugar would be the better choice to make if trying to reduce sugar intake. But what about other ingredients in soft and fruit drinks? There has been considerable concern raised recently about the amount of caffeine young children are consuming in cola drinks. Are you aware of the other ingredients and food additives that are in the soft and fruit drinks you are consuming? Can they also have the potential to affect your health in a negative way? This is a far less discussed concern than sugar, but perhaps we should be more aware of what we are consuming?

Table 1: Grams of sugar per 100g and per container size for the drinks analysed

Drinks analysed	Container size (ml)	Number of serves in container	Average sugar in 100ml	Average sugar in container	Kj per container
Berri—Apple Juice	400	2	10.3g	41.2g	720kj
Brownes—Choc Chill	600	1	8.5g	51.0g	1578kj
Brownes—Orange C	300	1	10.8g	32.4g	600kj
Coca Cola	390	1	10.6g	41.0g	702kj
Coca Cola—Zero	390	1	0g	0g	5.5kj
Coke Vanilla	600	1	10.9g	65.4g	1110kj
Diet Coke	600	3	0g	0g	9kj
Fanta—Orange Flavour	390	1.95	12.3g	48.0g	826kj
Fuze—Raspberry	600	3	11.0g	66.0g	1122kj
Invigoration V Energy Drink	250	1	11.5g	28.7g	465kj
Lipton Ice Tea—Peach Flavour	500	1	6.9g	34.6g	600kj
Masters—Mocha	300	1	10.3g	30.9g	1119kj
Play Sports Water—Raspberry	500	1	4.2g	21.0g	385kj
Powerade—Berry Ice	600	3	6.3g	37.8g	828kj
Red Bull Energy Drink	250	1	10.7g	26.7g	480kj
Ribena—Blackcurrant Fruit Drink	390	1	12.5g	41.3g	710kj
Supa Shake—Chocolate Honeycomb	500	2	12.5g	62.5g	2020kj
Supa Shake—Dome Cappuccino	500	2	10.9g	54.5g	2015kj

Table 2: Ingredients in the analysed drinks

	White ingredients— considered safe	Green additives— considered safe	Orange additives— consume moderately	Red additives— may be a hazard to health
Berri—Apple Juice (5)	reconstituted apple juice	vitamin C 300	food acid 330, flavour	natural colour 150d
Brownes—Choc Chill (10)	skim milk, whole milk, cocoa emulsifier (soy lecithin)	emulsifier 471	sugar, flavour, vegetable gum 412	vegetable gum 407, colour 155
Brownes—Orange C (8)	water, reconstituted orange juice	food acid 300, colour 160e	sugar, food acid 330, flavour, preservative 202	
Coca Cola—Regular (6)	carbonated water		sugar, food acid 338, flavour, caffeine	colour 150d
Coca Cola—Zero (10)	carbonated water		food acid 338, food acid 331, flavour, caffeine	colour 150d, sweetener 951, sweetener 950, preservative 211, contains phenylalanine
Coke—Vanilla (6)	carbonated water		sugar, food acid 338, flavour, caffeine	colour 150d
Diet Coke (10)	carbonated water		flavour, food acid 338, food acid 330, caffeine	colour 150d, sweetener 951, sweetener 950, preservative 211, contains phenylalanine
Fanta—Orange Flavour (8)	carbonated water, orange fruit from concentrate	antioxidant 300	sugar, flavour, food acid 330	preservative 211, colour 110
Fuze—Raspberry (7)	reconstituted fruit juices, natural flavour sourced from fruit, grape skin extract	vitamin C 300, dimethyl dicarbonate 242, carbon dioxide 290	citric acid 330	
Invigoration V Energy Drink (12)	carbonated water, guarana extract, inositol, vitamins—B3, pantothenic acid, B6, B2, B12	glucuronolactone	acidity regulator 330, acidity regulator 331, sugar, taurine, colour 150, caffeine, flavours	
Lipton Ice Tea—Peach Flavour (7)	water, tea extract, peach juice	antioxidant 300	sugar, food acid 330, flavours containing wheat derivative	
Masters—Mocha (9)	milk, milk solids non-fat, instant coffee, cocoa powder		sugar, flavours	vegetable gum 407, colour 155, colour 133
Play Sports Water—Raspberry (9)	purified water, concentrated fruit juices, electrolytes (sodium chloride)		fructose, electrolytes (calcium lactate), flavours, food acid 330	preservative 211, preservative 223
Powerrade—Berry Ice (10)	water, sodium chloride	tri-potassium phosphate	sucrose, maltodextrin, food acid 330, food acid 331, flavour, tri-potassium citrate	colour 129
Red Bull Energy Drink (11)	carbonated water, inositol, vitamins (niacinamide, pantothenic acid, B6, B12, riboflavin)	glucuronolactone	sucrose, taurine, glucose, acid 331, caffeine, colour 150, flavours	
Ribena—Blackcurrant Fruit Drink (6)	water, blackcurrant juice 5%	antioxidant 300	cane sugar, food acid 330	preservative 211
Supa Shake—Chocolate Honeycomb (15)	skim milk, whole milk, whey protein, chocolate, cocoa, malt powder	vegetable gum 412, vegetable gum 460,	sugar, glucose syrup, vegetable gum 466, flavour	vegetable gum 407, colour 102, colour 110
Supa Shake—Dome Cappuccino (12)	skim milk, whole milk, whey powder, coffee	vegetable gum 412, vegetable gum 460	glucose syrup, sugar, vegetable gum 466, colour 150, flavour	vegetable gum 407

Total number of ingredients is in brackets.

IS THERE A PROBLEM WITH SOME ADDITIVES?

There are hundreds of food additives allowed by the Food Standards Australia and New Zealand (FSANZ) for use in our foods. They have been tested before being accepted as safe. Additives can only be used in the foods that are listed for that additive. All additives must be listed as ingredients by their class name followed by the name and or number of the additive. For example, the yellow colour tartrazine could appear as Colour (Tartrazine) or Colour (102). Additives are intended to be used in very small quantities. Some additives have been linked to behavioural and other health problems in sensitive people. If your diet is largely made up of highly processed or convenience-type foods, with little variety, you may be consuming larger than intended amounts of these additives. Young children especially may consume inappropriate additives when they drink large amounts of cordials, soft drinks and flavoured milk drinks. Coca Cola has come under fire for the use of caffeine as a flavour enhancer (see Media Watch on page 9 of this issue).

Perhaps you could look at the additives in your favourite drinks and find out more about them. Ask yourself what role they have in the drink. Why do manufacturers need to use them?

The health of young people and the adults they will become is linked to the establishment of healthy behaviours in childhood. Risk factors such as excess weight gain, unhealthy dietary patterns and physical inactivity during childhood and adolescence can result in increased risk of developing cancer, cardiovascular disease, diabetes, hypertension, and osteoporosis later in life. Children who adopt healthy lifestyle habits by learning to make better choices, especially with food and drinks, are more likely to continue these behaviours throughout life.

RECOMMENDATIONS

We have a huge array of foods and drinks from which to choose. Those with a long storage life are mainly highly processed. Today the food and drinks we consume can detract from rather than add to our ongoing sense of wellbeing.

Therefore:

- eat mainly fresh food, in particular raw vegetables and fruits
- drink plenty of water
- avoid energy dense food and drinks such as those high in sugar and fats
- avoid artificial sweeteners as sugar alternatives
- avoid regularly consuming highly processed foods and drinks that contain additives
- balance energy intake with physical activity
- avoid excessive weight gain throughout the lifecycle
- finally, follow the recommendations of *The Australian Guide to Healthy Eating*.

STUDENT ACTIVITIES

1. The article outlines a list of 'problems(s) with soft and fruit drinks'. Identify three problems that are related to the:
 - a. nutritional value of the drinks
 - b. amount of drinks consumed

2.
 - a. Use the article and other sources to make a list of foods containing added sugar; include those that contain 'hidden sugars'.
 - b. Identify other names used for sugar in ingredients listed on labels.
3.
 - a. Provide some of the health reasons why sugar should be limited in the diet of children and adolescents.
 - b. Summarise the information presented in Table 1 by calculating the range and means of the values in each of the columns. (Consider whether or not you should include the Diet Coke and Coca Cola Zero values in your calculations of means).
 - c. Compare the range and mean for the last two columns with the recommendation for daily kilojoule (10,500kj) and sugar intake (less than 10% of kj which is equivalent to approximately 65 grams) for an average 16 year old female.
 - d. What conclusions can you draw?

Going Further

1.
 - a. Survey the beverage consumption in your year level or class. Ask the following questions: Which beverages do they drink? What quantity of each do they drink per week? Where are the drinks purchased? Where are they consumed (at home, school, other) and when? Collate and summarise your results. What comments can you make about your results?
 - b. Produce your own version of Table 1—showing only the drinks that are commonly consumed within your class.
 - c. As a class activity, produce your own posters of beverages, similar to those described in the article. Choose beverages that are commonly consumed at your school—from canteen, other outlets and home. Display your posters as part of an awareness campaign for staff and students at your school.

Web Activities

3. Use the Betterhealth website <www.betterhealth.vic.gov.au> or other reputable sites to research the health problems associated with over consumption of sugar for children and adolescents. Your teacher will provide a list of reputable websites.
4. Visit the Foods Standards Australia New Zealand (FSANZ) website <www.foodstandards.gov.au>. Click on 'Food Matters', then 'Food Additives'. Use this information to answer the following questions.
 - a. Choose 3 drinks commonly consumed in your class. List the additives from the labels of these drinks. For each of the additives listed, describe the most common reasons for its use in the drinks.
 - b. Using Table 2 in the article, and the last column choose 3 different additives that may be 'linked to health problems in sensitive people'. For each of the additives chosen:
 - i. Record its name and number
 - ii. Describe the type of additive and the reasons it would be added to foods.
 - iii. Outline the possible health problems associated with its use by people who have an intolerance to it. (You may need to use information from other websites – ask your teacher for a list of reputable websites).
5. Debate
In your class, debate the statement 'Today the food and drinks we consume can detract from rather than add to our ongoing sense of wellbeing.'

MEDIA WATCH

by Dorothy Carey

SUN EXPOSURE MESSAGE IS NOW SO CONFUSING

How much sun exposure is good or bad remains unknown and people should seek their doctor's advice on what is best for their individual circumstances, a new study has found.

A report in the latest edition of the Medical Journal of Australia says exposure to the sun is necessary for the production of vitamin D, which is important for healthy bones and may also help prevent several types of cancers.

But Australians are becoming more and more confused because they are getting mixed messages about keeping out of the sun to avoid skin cancer and getting enough sun to produce healthy levels of vitamin D, cancer researcher Rachel Neale said.

'It still may not be possible to provide a single message about healthy sun exposure appropriate for the whole of Australia,' said Dr. Neale, who is an epidemiologist with the Queensland Institute of Medical Research.

'It has been estimated that six to 12 minutes of winter sun exposure three to four times a week may be sufficient to produce healthy levels of vitamin D in Brisbane, compared with 51 minutes in Melbourne.'

She said that many factors contributed to a person's skin cancer risk or ability to synthesise vitamin D, including age, skin type, body mass index, food choices, liver health and environmental conditions.

'We don't yet know the minimum amount of sunlight needed to maintain healthy bones, let alone prevent internal cancers or chronic diseases,' she said.

National guidelines recommend sun protection be used if the

UV index is three or higher and that people at risk of skin cancer should use more rigorous sun protection.

The guidelines also suggest people living in southern Australia might need more sun exposure to achieve healthy vitamin D levels and that the elderly and those who cover themselves with clothing for cultural reasons might need vitamin D supplements. But Dr. Neale said further research was needed.

In the interim, sun-safe practices should be encouraged and supplements used where necessary until a basic understanding is achieved about the relationships between chronic disease, vitamin D and sunlight.

The West Australian, 15 January 2007

STUDENT ACTIVITIES

1. Explain the link between exposure to sun and nutrition.
2. Why is the sun exposure message now seen as 'confusing'?
3. a. List the factors that contribute to a person's ability to synthesise vitamin D.
b. Provide more information to explain each of these factors? Use resources where necessary.
4. Give three examples of groups of Australians who may be at risk of achieving enough sun exposure to provide for healthy vitamin D levels.

Going further

5. Complete an Internet search to find the link between sun exposure and vitamin D.
a. What factors should be considered in determining levels of sun exposure and vitamin D intake?
b. Select two of the Internet sites and summarise the findings from them.

COKE IN THE FIRING LINE AS CAFFEINE FLUNKS THE TASTE TEST

by Jill Stark

COCA-COLA has come under fire again for fuelling the childhood obesity crisis after Melbourne research found that adding caffeine — an addictive stimulant — does not enhance flavour.

Soft drink companies say caffeine adds flavour to cola, but a scientific taste test conducted by Deakin University found consumers could not tell the difference between caffeine-free Coke and a version with caffeine.

Head researcher Russell Keast said it was 'unethical' for companies to use caffeine if it did not enhance flavour and could lead to young people becoming addicted to sugary drinks.

Dr Keast, from Deakin's school of exercise and nutrition, has presented the findings to state and federal governments and is urging health ministers to consider an age restriction for caffeinated soft drinks.

'This is an issue that government should look at regulating, and maybe caffeinated soft drinks shouldn't be marketed to children at all,' he said.

'Children don't have the cognitive ability to understand why they may be getting moody or irritable because their caffeine high has waned over time and they're wanting more.'

'If it's there purely not as a flavouring but as an addictive agent or to promote caffeine dependence, then that would be unethical.'

In the study, outlined in the American journal *Appetite*, 30 people were given three small cups of commercial decaffeinated Coke.

Researchers spiked one sample with caffeine and asked

participants to spot the difference. Nobody could tell the difference in repeated tasting.

Coca-Cola was forced to defend itself in November when federal Health Minister Tony Abbott branded Coke as 'sugar-laden, caffeine-laden, acidic stuff' and urged the company to launch a marketing campaign warning of soft drink health risks.

The Age, Tuesday 9 January 2007. Reproduced by permission

STUDENT ACTIVITIES

1. According to the article, what is the relationship between Coca-Cola and childhood obesity?
2. Would the same relationship apply to adult obesity? Explain your response.
3. Describe the research process used in this study.
4. Explain the results of the study.
5. Apart from the relationship with obesity what other health problems are stated here as associated with caffeine intake in children?

Going Further

6. Soft drink intake has been associated for contributing to weakened bones, especially in females. Can you suggest a link here?
7. Research the use of caffeine in food. Why is it used? List some foods that contain caffeine.
8. a. Go to the following Internet site, <<http://www.betterhealth.vic.gov.au>>, do a search for caffeine and select the fact sheet.
b. Read the information provided. Discuss whether caffeine should be added to soft drinks.
9. Should children be protected from consuming caffeine? Justify your response. How could this be achieved?
10. Find out and make a list of other drinks that contain caffeine. Select one as an example and show how it is marketed.

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